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SOME BYGONE OPERATIONS IN SURGERY.

BY SIR D'ARCY POWER K.B.E., LONDON.

XI. THE REMOVAL OF A SEBACEOUS CYST FROM KING GEORGE IV.

SURGERY had made much progress between 1737, when Ranby operated upon Queen Caroline of Anspach for a strangulated umbilical hernia, and 1821, when Astley Cooper removed a sebaceous cyst from the head of King George IV. But the progress was not sufficient to make a surgical procedure safe, and Astley Cooper's own account of his trepidation affords interesting reading for a surgeon of the present day. He says:—

"The King sent to Sir Everard Home, myself and Brodie to go to Windsor to see a tumour on the summit of his head, which annoyed him from its appearance, and was growing larger. When we saw it, it was tender, painful and somewhat inflamed; and we thought it best to delay the operation. The King was much disappointed, but yielded to our advice.

"In the spring of the succeeding year, 1821, Sir Benjamin Bloomfield came for me to go to Brighton to see the King and I went down with Bloomfield. The King came into my room at one o'clock in the morning and said 'I am now ready to have it done. I wish you now to remove this thing from my head.' I said 'Sire—not for the world *now*—your life is too important to have so serious a thing done in a corner. Lady C—— S—— died of Erysipelas after such an operation and what would the world say if this were to be fatal? No, too much depends upon your Majesty to suffer me at one o'clock in the morning in a retired part of the Pavilion, to perform an operation which however trifling in general, might by possibility be followed by fatal consequences.' He hastily said, 'This is the second time I have been disappointed.' 'Yes, Sire, I am sorry for it, but I should not choose to do it, unless Sir E. Home, Mr. Cline and Mr. Brodie were present. Mr. Cline, the world would say, would not advise anything without due consideration and from my long knowledge of him, too, I should like him to assist me and he would not object to bear a part of the onus under any circumstances.' He said, 'Well, I respect Cline and I dare say he respects me, *although we do not set our horses together in politics.*' I replied, 'Perhaps not, your Majesty, but

the best policy, I think, will be to have him.' 'I will have it done as soon as I come to town then.' 'Very well, Sire,' and on the following morning at eleven o'clock, I returned to London.

"The King came to town soon afterwards and I went to the levée and he said, 'How do you do, Cooper?—well, next Tuesday.' I called upon Lord Liverpool and requested him to persuade the King to let Home do the operation, as that was the usual etiquette, he being Serjeant Surgeon. Lord Liverpool said that it was very difficult to interfere respecting the choice of a medical man. I was very averse from doing it; I had always been successful and I saw that the operation, if it were followed by erysipelas, would destroy all my happiness and blast my reputation. On the next day Home wrote to me that he should do the operation on Wednesday and I requested that Cline might be present to which Home consented. On the Wednesday we all met, Halford, Tierney, Home, Cline, Brodie and myself. Mr. Cline said 'Who is to do the operation?' I said, 'Sir Everard.' Soon afterwards Sir Henry Holland was called out of the room and almost immediately returning said to me, 'You are to do the operation.'

"I was thunderstruck and felt giddy at the idea of my fate hanging upon such an event. I said 'I have not come provided with instruments.' There was no time for parlance for the King directly entered the room and said 'Where am I to sit?' I replied 'here, Sire,' taking a chair to the window and begging an instrument of Home, I made an incision into the tumour and emptied it of its contents. Then I found it adhered strongly to the scalp and upon the side on which I stood which was about three-fourths of its size, I with difficulty detached it from the skin without cutting the skin itself. On that side on which Cline stood I begged him to detach it which he did but it took up a great deal of time on the whole. The edges of the wound were brought together and lint and plaister applied.

"The King bore the operation well, requested that there might be no hurry and when it was finished said 'What do you call the tumour?' I said 'A steatoma, Sire.' 'Then' said he 'I hope it will stay at home and not annoy me any more.'

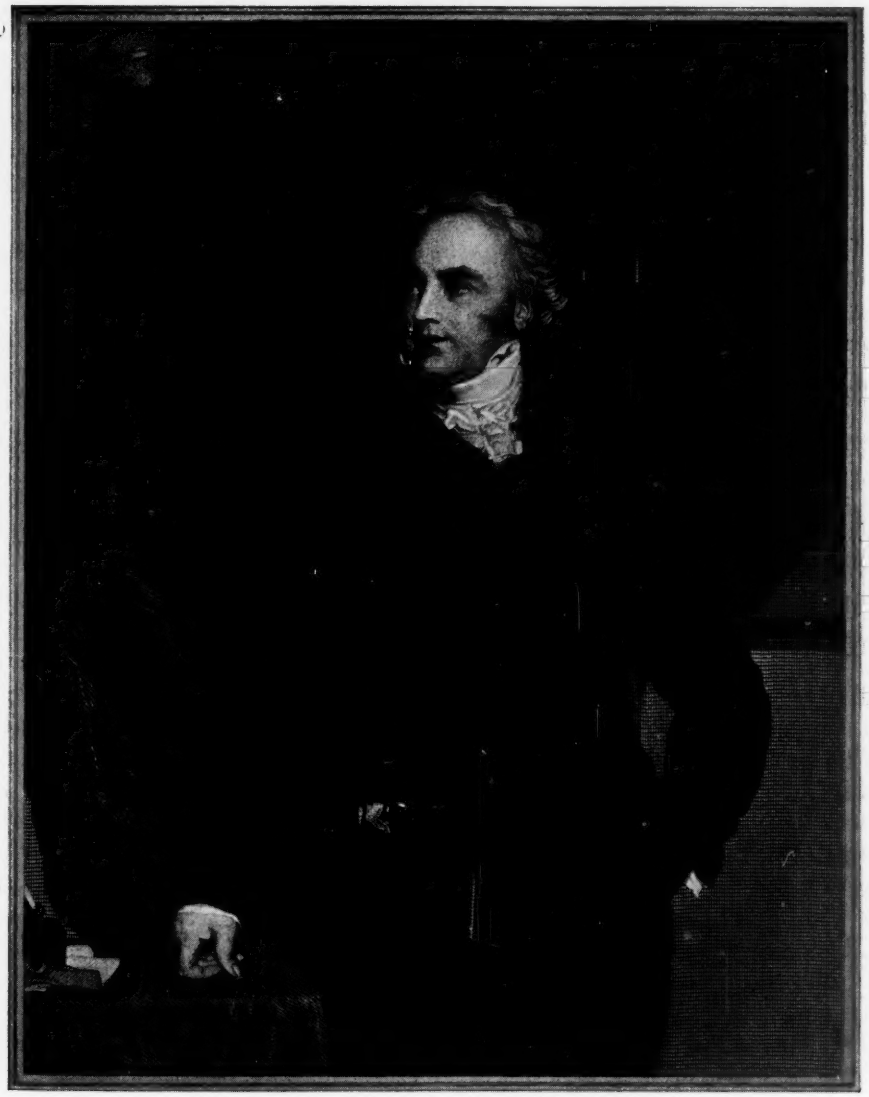
"The King went on well until Saturday; when he came in to us, he said 'I have not slept all night and I am damned bad this morning; my head is sore all over.' I immediately thought erysipelas was coming on and that we should lose him. I called in the middle of the day at Carlton Palace and again in the evening and he was much the same.

"The next day when I went the King was on the sofa—his great toe was red with gout—and his head had lost its soreness and all its unpleasant feelings. From this time the wound healed in the most favourable manner.

"In a fortnight afterwards he said 'Lord Liverpool has promised to make you a Baronet but I will not suffer it, I shall do it myself.' I thanked him and said 'Since your Majesty is so kind let me say, if it be not entailed upon my nephew Astley whom I have adopted and educated it will lose much of its value.' He immediately said 'It shall be made out as you wish.'

"He afterwards in six months sent me a beautiful épergne for which he gave the plan himself and which cost him five hundred guineas and we continued the best friends."

Three days after the operation Astley Cooper was sent for hurriedly to see the King and returned smiling and saying that it was a false alarm and added " but pray tell me do you see anything particular about me ? for the King did not seem in good tune ; he looked very hard at me from head to



SIR ASTLEY PASTON COOPER, Bart.

Reproduced from the Painting by Sir Joshua Reynolds.

foot and I cannot understand why—do you see anything ?—Why I said I should have put on a white cravat and a clean shirt or at least have washed my hands before I waited on his Majesty. The fact is that Sir Astley had performed a slight operation just before he went to the Palace by which some

blood had stained the sleeve of his shirt, where it projected from the wrist and his hands were also not perfectly free from it. Mr. Cooper then looking at what I had pointed out to him said " God bless me, so I ought but I was not aware of it—the King, Sir, is very particular—he was lying on a couch under a canopy with a red turban on his head and looked displeased—and now I see the reason of it." Dr. Badeley, who was then acting as Cooper's assistant, tells the story and Bransby Cooper reports it.

This account shows that there was a very real danger of the operation leading to some form of acute septic poisoning then summed up under the general term 'erysipelas'. It might have come from the surgeon because Astley Cooper considered any day wasted unless he had dissected for at least an hour, and this at a time when subjects were not injected and had often been obtained by resurrection methods. From the King because he was in poor health, very fat, and so plethoric that 130 oz. of blood had been taken from him to relieve an attack of suffocation from which he had suffered in the February of the preceding year.

Of the actors in the drama, Henry Cline, whose judgement was considered of such great importance, had been Astley Cooper's master at St. Thomas's Hospital. He was radical at a time when politics counted in everyday life. He was a devoted adherent of Horne Tooke, the agitator, and of John Thelwall, the reformer who used to cut the froth off a pot of porter and invoke a similar fate on all kings. Cline, too, was known to be in favour of the French Revolution. It was said on the other hand that his character was like that of Washington, for he devoted himself to what he considered to be to the advantage of his country and surrendered any distinction he might have attained when he had accomplished his object. He resigned his post of Surgeon to St. Thomas's Hospital in 1810, and in 1821 he was aged 71.

Astley Cooper, aged 53, was recognized by general consent as the greatest operating surgeon of his generation. He was Surgeon to Guy's Hospital and lectured at St. Thomas's, for the two hospitals were not separated until 1825.

Sir Everard Home, aged 65 in 1821, was the brother-in-law of John Hunter. He was Surgeon to St. George's Hospital and had been appointed Sergeant-Surgeon to the King in 1808. To salve his mortification at not being allowed to operate, his son, who was then a very young lieutenant in the Navy, was promoted out of his turn to the rank of Commander.

Benjamin Brodie was only at the beginning of his career, for he was not born until 1783. He had been house surgeon at St. George's Hospital under Home and had probably come with him to assist at the operation. It is evident, however, that his pleasant manners and gentle touch made a favourable impression upon the King, who afterwards consulted him frequently and appointed him Surgeon-in-Ordinary in 1828. He became Surgeon to St. George's Hospital in July, 1822, having already acted as Assistant Surgeon for fourteen years.

Sir Henry Halford was President of the College of Physicians from 1820 until 1844. He was a favourite with George III, who conferred a baronetcy upon him, and he attended George IV, William IV, and Queen Victoria. He is described as vain, cringing to superiors, and haughty to inferiors, but he was a good practical physician.

Sir Matthew Tierney was Physician-in-Ordinary to George IV. He was in practice at Brighton, and throughout his life was a strong supporter of vaccination. As a protégé of Lord Berkeley he had made Jenner's acquaintance when he was living at Berkeley Castle.

Lord Liverpool was Robert Banks Jenkinson (1770–1828), the second Earl of Liverpool, who was Prime Minister for fifteen years.

Sir Benjamin Bloomfield was the confidant of George IV and acted as his private secretary. He fell into disfavour in 1822, was sent as minister plenipotentiary to the Court of Stockholm, and was raised to the Irish peerage as Baron Bloomfield of Oakhampton and Tipperary in 1825. His social and musical attainments brought him to the notice of George IV, when he was living at Brighton doing duty with the 10th Hussars.

THE RELATIONSHIP OF THE STRUCTURE OF THE ENLARGED PROSTATE TO THE END-RESULTS OF PROSTATECTOMY.*

By E. W. RICHES,

ASSISTANT SURGEON AND ASSISTANT UROLOGIST TO THE MIDDLESEX HOSPITAL;

AND E. G. MUIR,

ASSISTANT PATHOLOGIST TO THE MIDDLESEX HOSPITAL; BERNHARD BARON RESEARCH SCHOLAR, ROYAL COLLEGE OF SURGEONS.

In assessing the prognosis and results of prostatectomy for enlarged prostate, attention has hitherto been confined mainly to two considerations, the personal factor of the patient and the type of operation performed. As regards the patient, the duration of symptoms and consequent degree of renal impairment appear to be of more importance than his actual age, and the presence of extra-urinary disturbances undoubtedly affects the final results in a series of cases. As regards the type of operation, discussion still continues on the relative merits of suprapubic, perineal, and perurethral prostatectomy, and of the open or closed operation in the first procedure. Many variations in after-treatment are also in use, and for each some benefit is claimed in reducing complications and improving results generally. One factor in prognosis which appears to have received insufficient consideration is the histology of the prostate removed. Cases of frank carcinoma, or those which prove to be malignant on microscopical examination, are notoriously difficult to remove and prone to recur; but even if these are excluded it is found that some prostates are enucleated easily and others with difficulty, so as to necessitate dissection, and there is a general impression that the former are more satisfactory in their end-results than the latter.[1] The present investigation has been undertaken in order to establish, if possible, statistical evidence of a correlation between the type of prostate, the symptoms produced, and the incidence of complications and ultimate prognosis after prostatectomy. For this purpose all cases of prostatectomy at the Middlesex Hospital from 1924 to 1931 have been reviewed. They number 114, excluding those of malignant disease of the prostate, and include the results of nine different surgeons. All the cases were submitted to suprapubic prostatectomy, and this is the one common factor; the majority were carried out by Freyer's technique, but in some the open operation of Thomson-Walker was performed; ligature of the vas was added in a few cases. The after-treatment given differed according to the practice of the individual surgeon. Despite these variations, the relative incidence of inflammatory complications was about the same

* From the Department of Urology and the Bland-Sutton Institute of Pathology, Middlesex Hospital, and the Royal College of Surgeons.

throughout the hospital, a fact which lends support to the view that in determining the subsequent course of the case there is some other factor concerned besides the technique employed in the operation and after-treatment. All the prostates have been examined microscopically, and as a result a histological classification is suggested.

ANATOMY.

A brief description of the more salient features of the anatomy of the prostate is germane at this point.

The prostate consists of 50 to 60 glands, each opening into the urethra, with duct, ductules, and acini: 12 to 14 of these glands lie in a loose stroma of their own, which radiates out from the posterior aspect of the urethra to join the capsule of the gland; these are called the *posterior lobe*—the 'true prostatic glands' of the Continental writers. They form the whole of the posterior surface and the greater part of the lateral surfaces of the prostate. Their ducts, larger and longer than the others, converge to open around the orifice of the utriculus masculinus.

The remaining prostatic glands lie in the dense muscular tissue around the urethra, and are called the *peri-urethral glands* or central glands (Albarran and Motz[2]). According to their situation they are divided into four groups or lobes as follows: Two *lateral* (14 to 16 in number), the ducts opening into the groove on either side of the crista urethralis; *middle* (8 to 10), opening into the urethra above the utriculus masculinus; and *anterior* (4 to 6), which open into the anterior wall of the urethra and are occasionally absent.

Albarran's glands, a few small glands opening into the posterior wall at the internal meatus, are often continuous below with the middle lobe.

While the lateral, middle, and anterior glands are continuous with each other, the posterior lobe is partially separated from them by the outermost layer of plain circular muscle, sometimes incomplete, which surrounds the urethral musculature and is continuous above with the vesical sphincter. The term 'peri-urethral glands' applies therefore to the lateral, middle, and anterior lobes, and the application of this name to small submucous crypts in the prostatic urethra is wrong and misleading.

PATHOLOGY.

Cases of non-malignant prostatic obstruction are clinically of three main types: (1) The typical 'enlarged prostate,' usually an easily enucleated tumour of some size; (2) The small 'fibrous prostate'; and (3) The prostate in which the presence of calculi is the predominant feature. Histologically there is some evidence that the two former are both examples of the same pathological condition—namely, benign hypertrophy—while the calculous prostate, unassociated with benign hypertrophy, is a separate entity. The large fibrous prostate described by some writers is probably an intermediate form between the glandular and fibrous varieties.[3]

1. **Benign Hypertrophy** (*Adenoma of Prostate, Hypertrophic Prostatitis*).—A variety of names has been applied to this condition, the characteristic feature of which is the presence of multiple small areas of glandular tissue

in the prostate, which by their growth compress the surrounding substance, giving the appearance of an encapsulated tumour. A collection of these nodules lying in the muscular stroma around the urethra forms the mass which is commonly called an 'adenoma' of the prostate (*Fig.* 195).*

FIG. 195.—Section immediately above utriculus masculinus. Both lateral lobes are enlarged by many glandular nodules, some cystic, causing urethral deformity. Posterior lobe compressed. Age 80.

Site.—In 59 prostates removed at autopsy in which benign hypertrophy was present, the areas involved were as follows :—

	PER CENT	
Lateral lobes	90	
Middle lobe (associated with lateral) ..	34	
Middle lobe (alone)	6	(All early cases)
Anterior lobe	1	
Posterior lobe	6	(Associated in every case with lateral-lobe changes)

(Albarran's glands are included with the middle lobe.)

Both the anterior lobe, which consists of only a few small glands, and the posterior, which forms more than half the bulk of the glandular tissue, are seldom involved.

Histology.—The microscopic appearance is familiar, and shows small, dilated, or cystic acini lying in a varying amount of fibromuscular stroma. The epithelium is that of the normal prostate, or in several layers with papillary processes. The acini may be empty, contain secretion, desquamated cells, or corpora amylacea. The stroma is commonly compressed and atrophic. Rarely muscular tissue may appear to form the greater part of the tumour,

* All the illustrations are from autopsy specimens except *Fig.* 210, which is from an operation specimen.

particularly if a section is examined from that part of the adenoma adjacent to the anterior urethral wall, where muscular tissue is abundant in the normal prostate.

The earliest stages in the formation of the small nodules or 'spheroids' can be seen in autopsy specimens. The first sign of their appearance is a group of acini around which the neighbouring tissues appear distorted or compressed (*Fig.* 196). The majority of the prostatic glands in which these early changes have been seen were normal except for somewhat exaggerated, thin papillary processes. In others this papillary hyperplasia starts in cystic acini, and here the histological appearance is similar to that described in the

FIG. 196.—Nodule showing compression of surrounding tissues simulating encapsulation. From the middle lobe. Age 63.

breast by Cheatle.[4] Adjacent papillæ come into contact, fuse, and give the appearance of another acinus.

Jacoby[5] believed from histological evidence that these acini retained communication with the urethra. Herring and Lawrence[6] attempted to demonstrate this fact by the injection of fusible metal. This method entails the subsequent destruction of the prostate, and no histological evidence can therefore be obtained. The difficulty is obviated by the use of Prussian blue in gelatin injected per urethram on autopsy specimens, and sections can subsequently be cut. These show that where the proliferating area is small, dye will frequently enter the acini (*Fig.* 197), though not when definite encapsulation is present. In the latter case this is probably due to the compression of the surrounding tissues and ducts.

Cysts in benign hypertrophy become increasingly frequent in the later decades (*see Figs.* 195, 199).

AGE	HISTOLOGICAL APPEARANCE OF ACINI IN 'SPHEROIDS'
40–49 50–59	.. Small acini, cysts rare.
60–69	.. Cysts in 40 per cent. In 5 per cent cysts predominated.
70 onwards	.. Cysts in 70 per cent. In 30 per cent cysts predominated.

The presence of cysts with incomplete septa and the fact that stages in their formation are frequently seen, such as the breaking down of walls between adjacent acini, suggest that cyst formation is the end-result of the epithelial proliferation, acini with desquamative changes representing a preliminary stage.

The presence of cystic acini was used by Ciechanowski[7] as evidence of duct stenosis with distension. Papin and Verliac[8] pointed out that while one part of the lining epithelium in a cyst might be flattened, in another high columnar cells might be present, and concluded that this appearance was

FIG. 197.—Dye injected from urethra into prostatic acini, in benign hypertrophy. Age 65.

not compatible with duct stenosis. Dye will enter these cystic acini with even greater ease than the small acini, and in the cases here examined no evidence of duct stenosis was found.

Posterior Lobe.—The changes found in the posterior lobe in cases of benign hypertrophy are usually described as those of 'compression atrophy'. That this is not the correct explanation is shown by the presence of similar atrophic changes in prostates without benign hypertrophy in men over 50.

BENIGN HYPERTROPHY IN LATERAL LOBES (Men over 50)	WITHOUT BENIGN HYPERTROPHY (Men over 50)
Atrophic acini present in posterior lobe in 77 per cent.	**Atrophic acini present in posterior lobe in 70 per cent.**
(In only 17 per cent of the above were the lateral lobes of sufficient size to have caused compression.)	

The changes seen in and around these atrophic acini are desquamation, fibrosis, and peri-acinar lymphocytosis. With the complete or partial shedding of the lining epithelium the corpora amylacea may act as foreign bodies and become surrounded by giant cells (*Fig.* 198). Cystic acini lined by only a trace of flattened cells are common, particularly on the lateral aspects of the prostate (*Fig.* 199). In 10 per cent of cases these cysts show the same papillary projections as those found in benign hypertrophy in the lateral lobes.

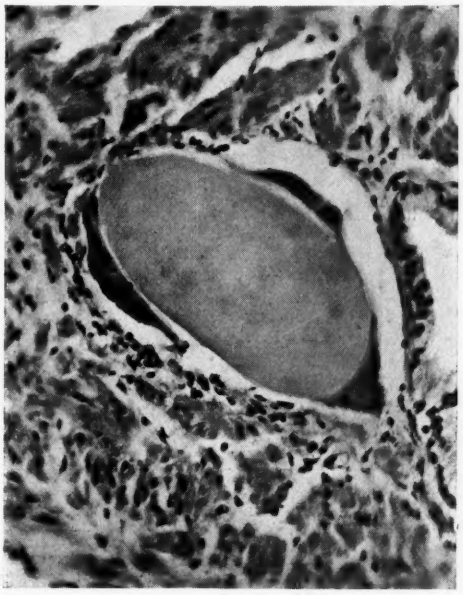


FIG. 198.—Atrophic acinus in posterior lobe showing loss of lining epithelium, peri-acinar lymphocytosis, and foreign-body giant cells applied to corpus amylaceum. Age 53.

Young[9] illustrates a case of an encapsulated adenoma in the posterior lobe. Adrion[10] believes that glandular proliferation may occur here, but that owing to the loose stroma it does not give rise to the encapsulated tumour seen in other parts. In this series of autopsy specimens the changes of benign hypertrophy were present in the posterior lobe in 6 per cent (*see Figs.* 206, 207).

The histological evidence suggests that atrophy in the posterior lobe apparently occurs earlier than in the rest of the prostate. In the sixth decade, when the changes of benign hypertrophy may already be present in the lateral or middle lobes, the posterior lobe is frequently partly atrophic. This early atrophy may be some explanation of the infrequency with which this lobe is involved by glandular proliferation.

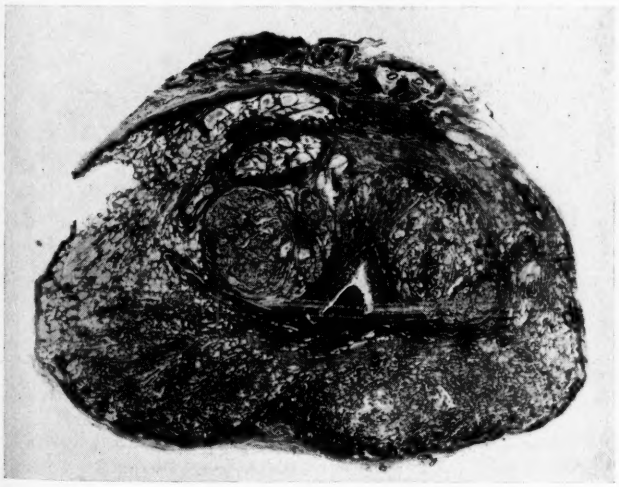


FIG. 199.—Nodules in both lateral lobes, some cystic. The lateral parts of the posterior lobe also show cystic changes, but posteriorly the acini are closely packed. Age 79.

The frequency of benign hypertrophy (88 per cent in men over 70 at autopsy) and its histology suggest an involutionary change common to the majority of men—a hyperplasia of some of the prostatic glands rather than a true neoplasm, which is followed in extreme old age by cyst formation.

2. **The Fibrous Prostate.**—Fibrous areas containing a varying amount of muscular tissue are frequently seen in the prostatic 'adenoma', and in a few cases constitute the bulk of the tumour. Their presence has led a number of authors to classify the tumour as a fibro-adenoma, or fibro-myo-adenoma,

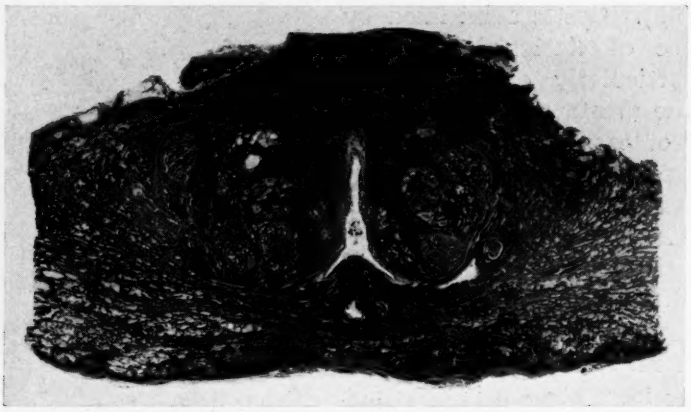


FIG. 200.—Section above utriculus masculinus. Nodules in lateral lobes. Age 68.

and they have been attributed either to a true neoplastic growth of the stroma or to an inflammatory change. The first view was that of Horn and Orator,[11] who believed that true myomata also occurred. Large areas of muscular tissue are not infrequently found in an 'adenoma', but in the normal prostate

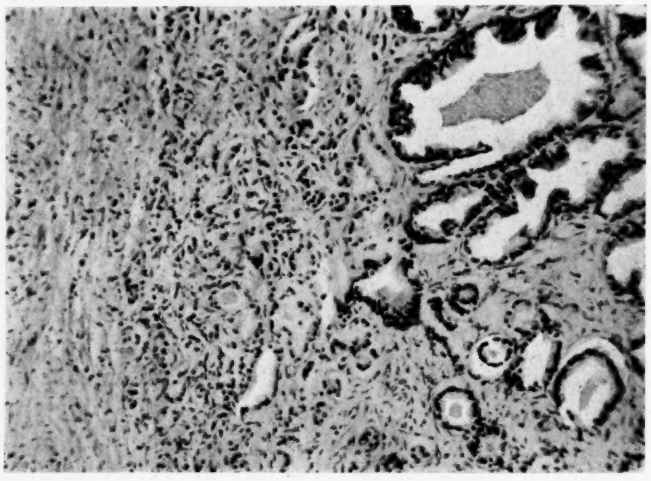


FIG. 201.—Edge of nodule from *Fig.* 200, showing replacement of peripheral acini by connective tissue. The outline of the original nodule can still be seen.

many bands of plain muscle are present around the urethra, the amount varying in individuals, and at different levels in the same individual. Young,[12] Adrion,[13] and others believe that the connective-tissue overgrowth is the result of infection, and that epithelial elements are frequently present in these

fibrous areas. Walker[14] states that the fibrous prostate represents an essentially different pathological condition from glandular hypertrophy.

A number of prostates removed at autopsy and by prostatectomy showed these fibrous nodules, and in many cases stages in their formation could be

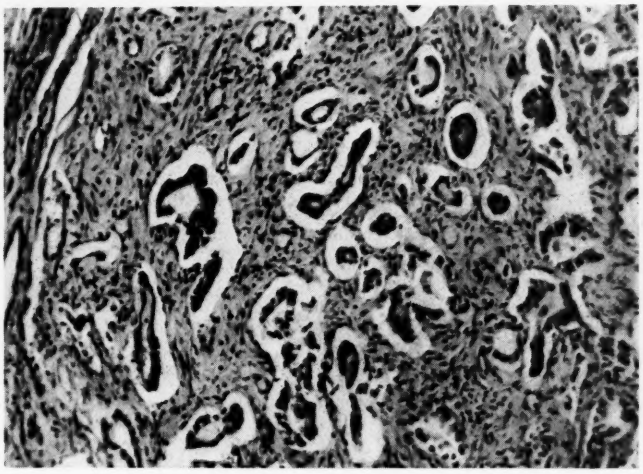


FIG. 202.—Further stage in the process of fibrosis. Age 61.

seen. A cellular fibrosis occurs around a glandular nodule, involving first the peripheral acini, which become compressed and finally obliterated. A fibrous nodule is left containing a number of arteriosclerotic vessels and

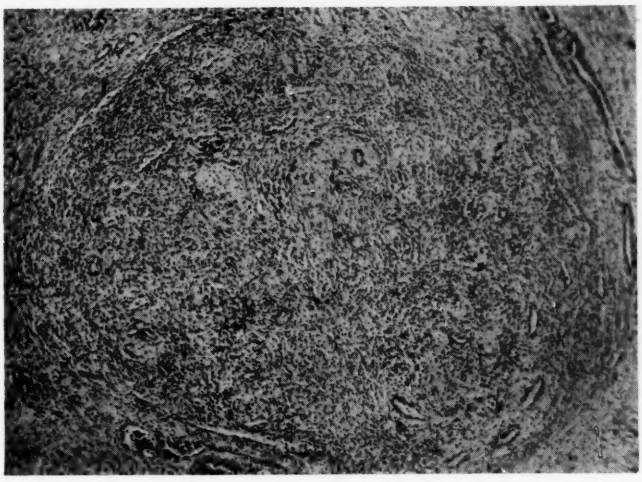


FIG. 203.—Complete replacement of glandular nodule by fibrous tissue. A number of arteriosclerotic vessels are present and the original capsule can still be seen. Age 65.

degenerate muscle fibres. The outline of the original glandular area still remains, though it no longer has such an encapsulated appearance (*Figs.* 200–203). The majority of these areas are commonly found in close proximity to the urethra, some covered only by mucosa, and may involve its whole length.

Overgrowth of connective tissue also gives rise to intra-acinar and intra-ductal papillomata with a thick connective-tissue stalk unlike the fine papillary processes usual in benign hypertrophy. They bear a close resemblance to the 'multiradicular' papillomata described by Cheatle[15] in the

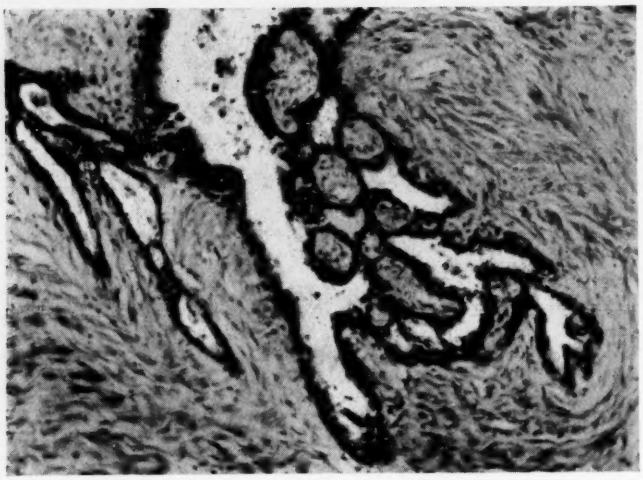


FIG. 204.—Commencing fusion of papillomatous processes in fibrosis.

breast, occur most frequently in the neighbourhood of the urethra, and by the fusion of opposing papillomata may cause complete obliteration of the acinus (*Figs.* 204, 205).

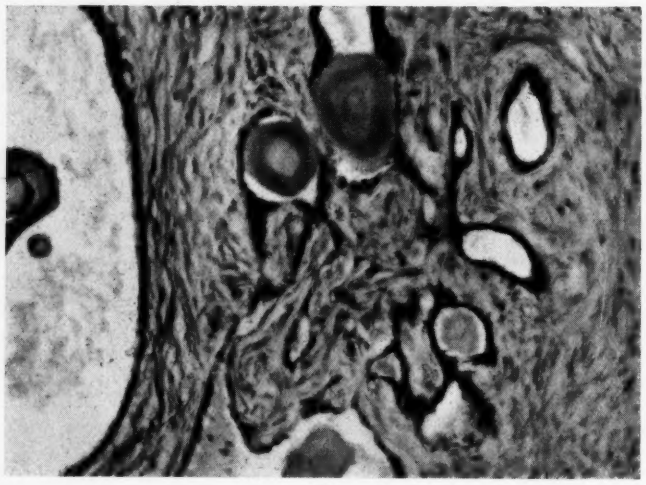


FIG. 205.—Later stage of fusion from the same case as *Fig.* 204, showing more complete obliteration of acini.

Where fibrosis is marked, all the glandular tissue in proximity to the urethra is replaced by fibrous nodules. The muscular tissues in which the glands lie are also affected, and the urethra is 'walled' by fibrous tissue containing traces of glands and degenerate muscle. The involvement of Albarran's

glands may cause stenosis of the internal meatus, or a fibrous 'bar' at the bladder neck (*Figs.* 206, 208). In the presence of such fibrosis it is apparent that any glandular mass present will be exceedingly difficult to enucleate.

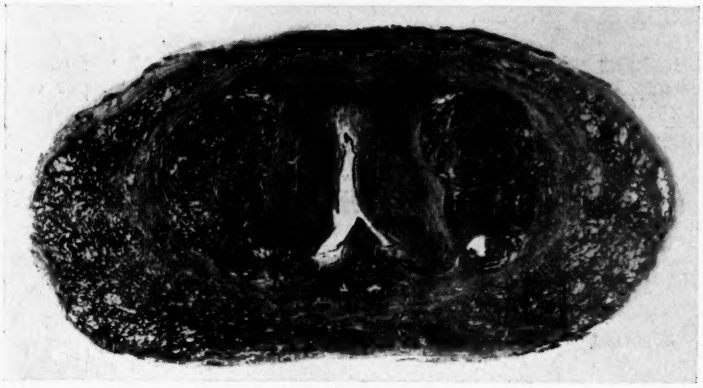


FIG. 206.—Glandular nodules in lateral lobes replaced near the urethra by fibrous tissue, in which the outline of some nodules is still visible. A small glandular nodule is also present in the posterior lobe. Age 68.

Thomson-Walker,[16] discussing the clinical conception of the 'fibrous' prostate, included in this term those prostates of 20 grm. or under. His figures give 3 per cent of prostates as showing hyperplasia of the stroma. In 5 per cent of the prostatectomies at the Middlesex Hospital the 'prostate'

FIG. 207.—The nodule in the posterior lobe outlined in *Fig.* 206. Atrophy of the surrounding acini is shown.

removed consisted mainly of fibrous or fibromyomatous tissue. Fibrosis was present though not predominant in 29 per cent. In those prostates removed at autopsy this fibrosis was present in :—

AGE	PER CENT
50–59	10
60–69	30
70 onwards ..	40

In two cases (2 per cent of prostates removed from men over 50) the change was sufficiently marked to classify them as 'fibrous' prostates, in both of which vesical trabeculation was present though there was no history of urinary symptoms.

The relation of a previous history of gonorrhœa is shown below.

Prostates with history of gonorrhœa, fibrosis in 47 per cent.
Prostates without history of gonorrhœa, fibrosis in 26 per cent.

Of 6 cases of prostatectomy in which the bulk of the prostate removed was fibrous, 2 had a history of gonorrhœa, one with stricture, and a third had chronic cystitis of many years' duration. These figures show that fibrosis

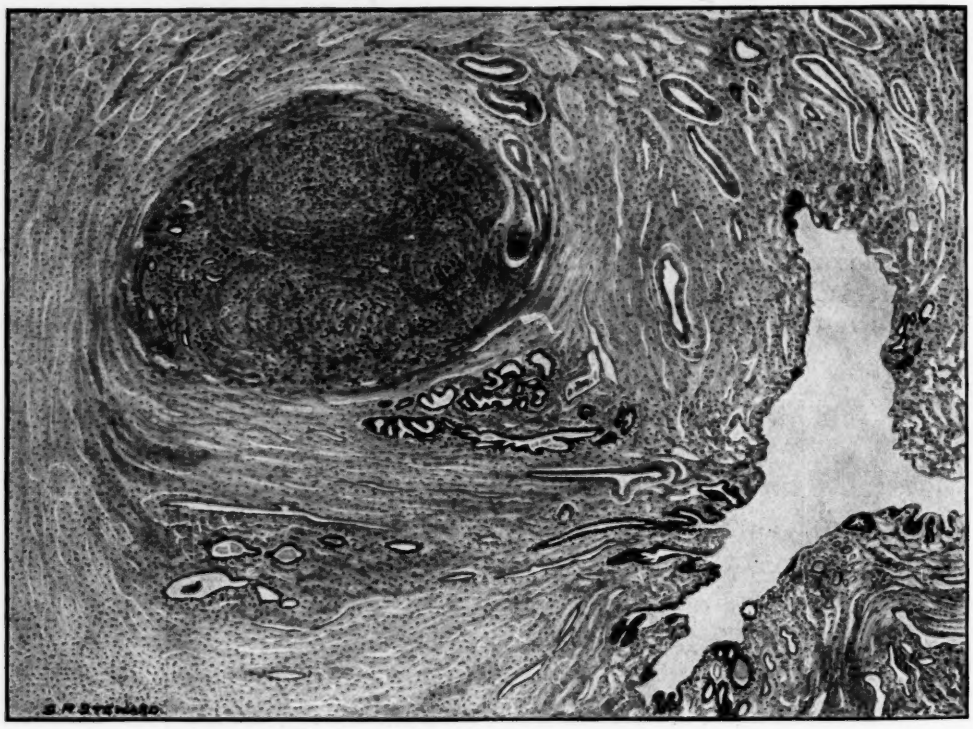


FIG. 208.—Fibrous nodule in Albarran's glands. From the bladder neck in the same case as *Fig.* 206.

is more common in, though by no means limited to, those cases in which a history of gonorrhœa is present. The absence of a history of gonorrhœa does not, however, exclude infection of some kind, nor does its presence necessarily indicate a previous prostatitis.

Acute inflammatory changes are present in the majority of prostates removed, particularly in that part of the 'adenoma' in close proximity to the urethra, and may be justifiably attributed to previous instrumentation. In 'fibrous' prostates chronic inflammatory changes are commonly marked, the perivascular lymphatics contain lymphocytes (*Fig.* 209), and the surrounding musculature is fibrotic. The other factor which bears an apparent relation

to fibrosis is age; whilst it is found in 10 per cent of prostates in the 6th decade, it is present in 40 per cent of those in the 8th.

The bulk of the evidence suggests that in a fibrous prostate we are not dealing with a separate pathological entity, but with a gland in which the

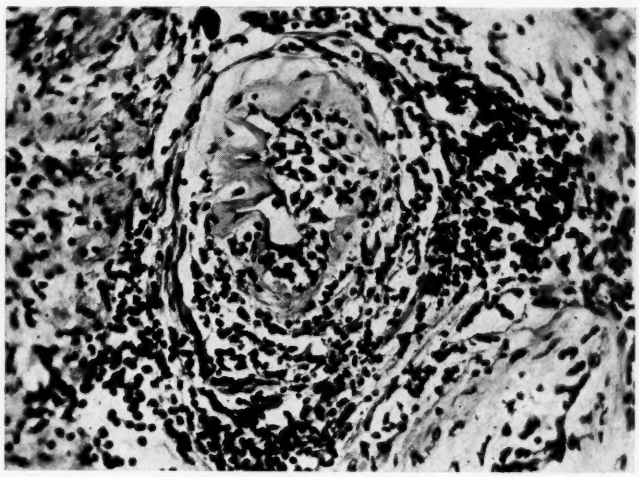


FIG. 209.—From a fibrous prostate, showing perivascular lymphocytosis. Age 61.

typical changes of benign hypertrophy are modified by fibrosis, due to chronic inflammation or to a degenerative change.

3. **Calculous Prostatitis.**—Small, gritty particles rarely larger than a pin's head are found in 40 per cent of prostates over 50. They are due to the deposit of mineral salts around corpora amylacea and in only a small number

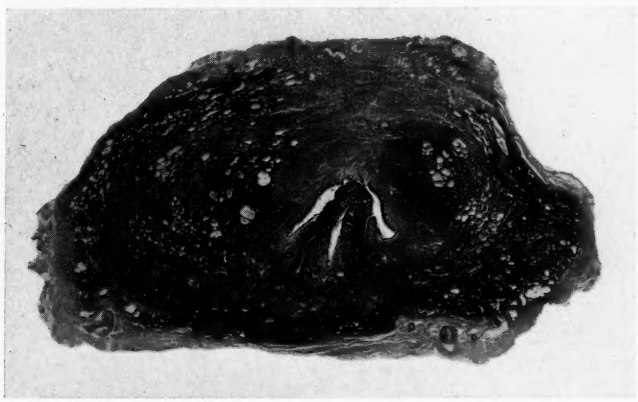


FIG. 210.—Section of calculous prostatitis. The atrophic appearance and the absence of prominent muscle bands is shown. Age 50.

are associated with inflammatory conditions. When inflammation is present the calculi are usually of larger size. Larger calculi, from 2 to 4 mm., occur in two conditions. In benign hypertrophy the ducts of the posterior lobe may contain calculi up to 1 to 4 mm. in diameter. These ducts as they pass

to the urethra occupy a horseshoe-shaped area behind the lateral lobes, and when these are enlarged in benign hypertrophy the calculi, if present, form a rim along the posterior aspect of the adenoma. Pressure on the ducts by the enlarging lateral lobes is a possible explanation of their presence.

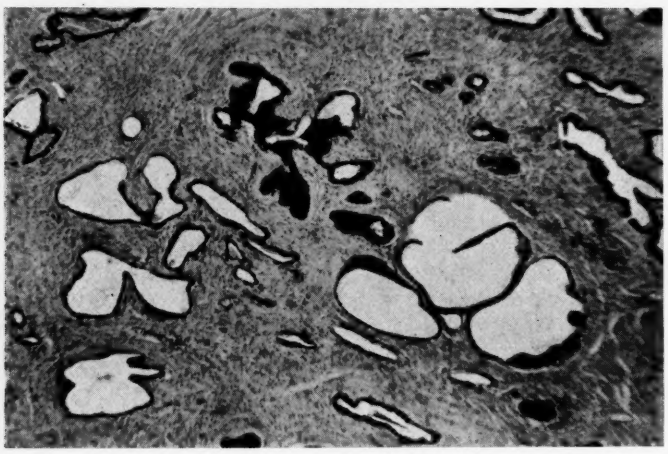


FIG. 211.—Same case as *Fig.* 210. Some acini are atrophic while others show epithelial hyperplasia. Many of the ducts and acini contained pus cells.

The term 'calculous prostatitis' is, however, restricted to a definite type of prostate which was present in 5 per cent of autopsy specimens. Macroscopically the prostate is smaller than normal. In the specimen illustrated,

FIG. 212.—Calculi in a duct in calculous prostatitis. Age 58.

from a man aged 50, the size was equivalent to that of a boy of 15 (*Figs.* 210, 211). On section dark particles of varying size are scattered throughout the gland, with larger calculi in the neighbourhood of the urethra (*Fig.* 212). Microscopically the ducts and acini contain pus, and periductal lymphocytosis

and fibrosis are marked. The latter extends to the greater part of the stroma and musculature.

Gland destruction may be marked; the acini lose their high columnar epithelium and may show areas of proliferating darkly staining cells (*Fig.* 211). Krompecher[17] believed that these represented an inflammatory hyperplasia of the supporting cells of the epithelium and that the rare squamous carcinoma of the prostate arose in such areas. The histological picture is that of an atrophic gland, the result of chronic inflammatory changes, associated with calculi which are probably secondary to the infection (prostatitis, urethritis, stricture). Unlike benign hypertrophy, these changes are not localized to any one site but are present throughout the *whole* gland.

Of 14 specimens examined (autopsy and biopsy) benign hypertrophy was present in only 1, and here in only a small area. The relative absence of such changes can probably be attributed to the destruction of gland tissue which occurs.

CLASSIFICATION.

The material removed by suprapubic prostatectomy in the 114 cases under review has been classified into four groups, on the lines of the preceding description.

Benign hypertrophy
- *A.* *Glandular*, where the material removed consists of glandular tissue in its normal stroma.
- *B.* *Intermediate*, or mixed type where traces of fibrous overgrowth are present with glandular tissue.
- *C.* *Fibrous*, composed almost entirely of fibrous tissue.

D. *Calculous prostatitis.*

The frequency of the four types was as follows:—

	Per cent
Glandular	57·5
Intermediate	28·5
Fibrous	6·0
Calculous prostatitis	8·0

CLINICAL INVESTIGATION.

For the clinical part of the investigation as many of the patients who were able to come have been interviewed and examined; others not able to come have filled in answers to a questionnaire. For those with whom contact could not be made recourse was had to the case notes alone. In this way the record of symptoms is based on 102 cases, that of mortality and post-operative inflammatory complications whilst in hospital on all the 114 cases, and the assessment of clinical results is made in respect of 68 cases who were either examined personally or who replied to the questionnaire.

Symptomatology.—The symptoms complained of by the patient, and their duration before seeking medical advice, are known in 102 cases comprising all four groups. In order of frequency they were as follows:—

	Per cent
Increased frequency of micturition	82
Difficulty in micturition	58
Pain on micturition	37
Acute retention of urine	34
Hæmaturia	11

Other symptoms, such as incontinence, alterations in stream, and dribbling at the end of the act amounted to less than 10 per cent of the whole, and have not been included.

The average duration of symptoms before seeking advice in all cases was 3 years 2 months. In a number of cases, however, the first symptom of which the patient complained was an attack of retention of urine, so that its duration was only a matter of a few hours, whilst the longest period in which symptoms were present was 14 years; thus a figure representing an average duration for the series is of little value. The average age of the patients on seeking advice was 62 years 6 months.

When the types are considered separately the frequency of the different symptoms is as follows:—

Symptom	Type of Prostate			
	Glandular	Intermediate	Fibrous	Calculous
	Per cent	Per cent	Per cent	Per cent
Increased frequency ..	67	74	100	87
Difficulty	60	55	53	62
Pain	29	23	33	62
Acute retention	48	41	33	12
Hæmaturia	20	13	0	12
	Years	Years	Years	Years
Average duration of symptoms	2	2¾	5½	2¾
Average age	63	65	64	58

Increased frequency of micturition was a constant feature in the fibrous type, in which type also there was a longer average duration of symptoms; this type, indeed, produces a picture somewhat resembling that of urethral stricture, the symptoms being due not to a glandular tumour, but to a progressive fibrosis surrounding the urethra and involving the muscles.

Difficulty in the act was present to about the same extent in each type, but pain was a more prominent feature in the calculous group. Acute retention was most common in the glandular and intermediate types, and in more than half of the cases of these groups in which it occurred there was more than one attack, relief by catheterization being followed by intervening periods of comparatively easy micturition. In the fibrous and calculous types, on the other hand, when acute retention did occur one attack was the rule, operation being necessary for its relief. In accordance with expectations hæmaturia was commonest in the glandular type, and was absent altogether in the fibrous prostates.

Post-operative Inflammatory Complications. — Under this heading are included, in order of frequency, epididymitis, secondary hæmorrhage, and ascending renal infection. The total of such complications was 34, an incidence of 29 per cent for the series. When the different histological types are considered separately the incidence of complications is seen to be relatively low in the glandular type, but considerable in the other three, with a gradual increase from the intermediate group to the cases of calculous prostatitis.

Type	Total	Complications
		Per cent
Glandular	66	16
Intermediate ..	33	42
Fibrous	6	50
Calculous	9	66

Considered in more detail the inflammatory complications were as follows :—

Glandular.—

Epididymitis	7	
Secondary hæmorrhage ..	2	
Ascending infection	2	
Total ..	11	out of 66 (16 per cent)

Intermediate.—

Epididymitis	9	
Secondary hæmorrhage ..	4	
Ascending infection	1	
Total ..	14	out of 33 (42 per cent)

Fibrous.—

Epididymitis	3	
Secondary hæmorrhage ..	0	
Ascending infection	0	
Total ..	3	out of 6 (50 per cent)

Calculous Prostatitis.—

Epididymitis	4	
Secondary hæmorrhage ..	(1)	occurring in one of the cases with epididymitis
Ascending infection	2	
Total ..	6	out of 9 (66 per cent)

Whilst the total number of cases is small, particularly in the fibrous and calculous types, the difference in incidence of complications is striking.

Epididymitis occurred either on one or both sides in 23 cases, or 20 per cent of the total. This figure approximates to that given by Randall[18] of 23 per cent, but is considerably less than that of Winsbury-White[19] (82 per cent), which, however, included the very mildest forms. It is sufficiently high to make division and ligature of the vasa a desirable addition to the operation, as advised by Morson.[20] That ligature alone is not sufficient was shown in one case in this series where epididymitis followed on the side ligatured but not on the side divided. The time of onset varied from a few days after operation to some weeks after leaving hospital; one case went on to abscess formation, and the remainder subsided under expectant treatment.

Secondary hæmorrhage occurred in 7 cases (6 per cent). It usually started about ten days after operation, and was responsible for two deaths. Ascending infection leading to pyelonephritis was the least common but most fatal complication; it occurred in 5 cases (4·4 per cent) and caused death in each instance. The possibility of the occurrence of minor degrees of ascending infection which did not prove fatal cannot be excluded.

The relationship between the histological type and the incidence of these complications is probably indirect, depending in part on the greater trauma inflicted in attempting to remove a fibrous or calculous prostate than one which is purely glandular, and in part on the lighting up of a previous chronic infection in the prostate.

Mortality.—The number of deaths from the operation was 13 (12 per cent), which compares favourably with the figure of 19·5 per cent for twelve general hospitals quoted by Thomson-Walker.[21] Figures are given for each histological type, but the numbers are too small to warrant any general conclusions.

Type	Mortality
Glandular	7 out of 66 (10 per cent)
Intermediate	4 out of 33 (11 per cent)
Fibrous	0 out of 6 (0 per cent)
Calculous prostatitis ..	2 out of 9 (22 per cent)

The cause of death was as follows :—

Glandular (7 cases)	Pyelonephritis, uræmia ..	2
	Primary hæmorrhage ..	2
	Secondary hæmorrhage ..	1
	Pulmonary embolus ..	1
	Myocardial degeneration ..	1
Intermediate (4 cases)	Pyelonephritis, uræmia ..	1
	Primary hæmorrhage ..	1
	Secondary hæmorrhage ..	1
	Syncope	1
Fibrous	.. ..	0
Calculous prostatitis (2 cases) ..	Pyelonephritis, uræmia ..	2

Fatal primary hæmorrhage thus only occurred after prostates with glandular elements had been removed, but not in the fibrous and calculous varieties. It is recognized that death is as a rule due to a combination of causes in these cases, and the discrepancies in different mortality tables have already been pointed out by Walker.[22]

Clinical Results.—The assessment of the results of the operation has been arrived at by a consideration of symptoms and by a complete examination of the patient whenever possible. From the symptomatic point of view the operation has been considered completely successful only if the patient was living in entire comfort as regards his urinary function, soundly healed, with complete control, able to pass a good stream without pain or difficulty, and not having to micturate more than twice during the night. Such cases are classified as 'normal', and on this basis the percentage of normal results in each of the four histological groups is found to be as follows :—

Type	Normal Results after operation
Glandular	29 out of 33 (88 per cent)
Intermediate	16 out of 22 (73 per cent)
Fibrous	1 out of 6 (17 per cent)
Calculous prostatitis ..	3 out of 7 (43 per cent)

Allowance must be made for the necessarily small number of cases of fibrous prostate and calculous prostatitis, but in this series it appears that a normal result after operation is five times as common in the glandular type as in the fibrous type.

The results in each group will now be considered in more detail.

Glandular Type (33 cases reviewed)—

Normal result	in 29	(88 per cent)
Increased frequency	in 3	
Nocturnal incontinence	in 1	

In general these patients were very well, and extremely pleased with the result of the operation. In three-quarters of the cases examined post-operatively the prostate was appreciable and showed adenomata on cystoscopy and rectal examination, and mild cystitis with some trabeculation was present in half the cases, although in the majority the urine contained no pus and was sterile on culture. In one the prostatic enlargement was considerable, although the patient had no symptoms. One patient had a return of difficulty in starting and frequency of micturition a year after operation, and it was found at this time that there was stenosis of the internal urinary meatus, so that only a filiform bougie could be passed. Gradual dilatation restored his function to normal, and cystoscopy showed irregularity of the prostatic margin. Of the three patients with increased frequency of micturition none had to pass water more than thrice in the night; one of these was a man of 76 who had had a large diverticulum of the bladder containing eighty-nine stones; this was excised seven weeks before prostatectomy was undertaken. One patient, a man of 75, found he could not hold his water by night, and therefore wore a collecting apparatus. The highest amount of residual urine was 3 oz.

Intermediate Type (22 cases reviewed)—

Normal result	in 16	(73 per cent)
Increased frequency	in 5	
Permanent suprapubic fistula	in 1	

In general the results were again good in this series. Prostatic adenomata were present on rectal and cystoscopic examination in half the cases examined, and some degree of cystitis in more than half, with some pus cells and *B. coli* in the urine. In one case the prostatic enlargement was considerable. The degree of increased frequency of micturition in the cases enumerated was higher than in the purely glandular type. There was stenosis at the internal meatus in two cases; in one of these the passage of the cystoscope caused the rupture of a band at the meatus, and his frequency of micturition was subsequently reduced from nine times at night to once, starting the act being also made much more easy. In the other, a frail man of 76, in whom out-patient instrumentation was not considered desirable, there was sufficient stenosis to produce a permanent suprapubic fistula for which an apparatus was worn. The highest amount of residual urine was $3\frac{1}{2}$ oz.

Fibrous Type (6 cases reviewed)—

Normal result	in 1	(17 per cent)
Increased frequency	in 2	
Delayed healing	in 2	
Sensation of incomplete emptying	in 1	

The final result in these cases was more satisfactory than the figures suggest, but only one could be classed as normal, and in this case there was

considerable scarring and injection around the internal meatus. In general the degree of cystitis present was more severe than in the previous groups, and in one case there was marked cystitis cystica around the internal meatus, which probably accounted for his sensation of incomplete emptying of the bladder. There had been sufficient stenosis of the internal meatus to delay healing for more than six months in two cases, and in a third the regular passage of bougies had been necessary for some weeks after operation. On rectal examination the prostate in all these cases was either flat or concave, and there was no evidence of new adenomatous formation. The highest residual urine was 1 oz.

Calculous Prostatitis (7 cases reviewed)—

Normal result	in 3 (43 per cent)
Increased frequency	in 2
Nocturnal incontinence	in 1
Death elsewhere two years after operation*	in 1

*Inquiry elicited the information that this patient had obstruction at the internal meatus.

The final results in this group were numerically slightly better than in the purely fibrous type, but residual cystitis was rather severe in some cases. One patient who was getting up four times at night to micturate had a persistent epididymitis five months after operation and his condition at that time was rather worse than before operation; one had residual calculi. The patient with nocturnal incontinence had a very severe cystitis, the infecting organism being *B. pyocyaneus*; this organism was present before operation in the bladder urine. Stenosis of the internal meatus of some degree was present in two cases, and the presence of adenomata was noted in only one, and these were small and in the anterior lobe. By rectal examination the prostates were appreciable as flat or apparently normal prostates and in no case felt enlarged. The highest residual urine was $2\frac{1}{2}$ oz.

DISCUSSION.

Examination of these cases showed that the final result was very good in those cases with purely glandular prostates, and good in the intermediate type, whilst in the fibrous and calculous cases it was much less satisfactory. The factors causing the poor results in the two latter groups were an increased liability to stenosis of the internal meatus and a more frequent and severe residual cystitis; these may both be considered inevitable results of the difficulty in removing all the fibrous tissue which commonly extends throughout the prostatic urethra in the fibrous type, and the impossibility of taking away all the infected gland in calculous prostatitis.

Re-appearance of the Prostate.—On the other hand, the re-appearance of the prostate was more marked in the glandular and intermediate types, and in two of these there was sufficient enlargement to suggest that further obstructive symptoms might ensue and necessitate a second prostatectomy. In the fibrous and calculous types post-operative rectal examination showed a flat or concave surface to the examining finger, and cystoscopy showed no adenomatous formation except in the one case already mentioned with small anterior adenomata.

Davies and Loughnane[23] found that of 19 prostatectomized patients examined with the urethroscope only 2 still showed total absence of the prostate. In one of these it is recorded that there was no prostatic enlargement felt per rectum before operation, from which it appears a possible conclusion that the case was one of fibrous prostate; in the other case no note is given of a pre-operative rectal examination.

Amount of Prostate removed at Operation.—It has already been shown that the posterior lobe is but rarely involved in the glandular changes of benign hypertrophy, but is subject to early atrophy and compression in lateral-lobe enlargement, and that it is partially separated from the remaining lobes by a layer of plain circular muscle continuous above with the vesical sphincter. This supports the view first advanced by Wallace[24] that suprapubic prostatectomy in the glandular or early intermediate types really consists of enucleation of the lateral, middle, and anterior lobes only, i.e., the peri-urethral glands as previously defined. The posterior lobe is probably only removed, and then only partially, when there is sufficient fibrosis to destroy the natural plane of separation between it and the remaining lobes, as may occur in the purely fibrous and calculous types.

Other Methods of Treatment.—In the glandular and intermediate types of benign hypertrophy the results in this series are in accord with the general conclusion of Walker that some 90 per cent of patients will survive the operation of suprapubic prostatectomy; complete urinary relief may be expected in from 70 to 90 per cent of cases. There remain the cases of fibrous prostate and calculous prostatitis in which some other procedure might be considered. The possible alternatives to suprapubic prostatectomy appear to be:—

1. *Catheter Life.*—This has many well-recognized disadvantages and should be necessary only in a small and decreasing number of cases.

2. *Regular Dilatation.*—This is of service in a limited number of cases, particularly of fibrous prostate.

3. *Perineal Prostatectomy.*—This has never been widely practised in England, and even in America its popularity seems to be on the wane.

4. *Per-urethral Partial Prostatectomy.*—If the results already claimed for it are borne out by those of larger series of cases[25] this appears to be the most hopeful procedure, certainly for the fibrous prostate and possibly for calculous prostatitis.

Recognition of Cases.—The recognition of the type of prostate encountered is thus of absolute importance; fibrous and calculous cases present a clinical picture with many common features, and in the latter decisive confirmatory evidence may be obtained by radiological examination or, as sometimes happens, by the striking of a loose calculus on instrumentation. The main diagnostic features of the fibrous prostate are discovered from the history, symptoms, and physical and endoscopic examinations, and the outstanding points as compared with glandular prostates, are here summarized:—

History.—Relatively long duration of symptoms; possible previous infection, e.g., gonococcal.

Symptoms.—Increased frequency of micturition the rule, hæmaturia the exception. Previous attacks of acute retention uncommon.

Rectal Examination.—Little enlargement of the prostate; induration without infiltration. Examination with a bougie in the urethra helps one to distinguish the induration of a fibrous prostate from the elasticity of a glandular enlargement on the one hand and the stony hardness of a carcinoma on the other.

Cystoscopy and Urethroscopy.—Some resistance to the introduction of the instrument in the prostatic urethra; little intravesical projection of the prostate, but possibly a small projecting collar, raised more posteriorly than anteriorly, or a posterior bar; cystitis and trabeculation; prostatic urethra may be shortened, and does not show the projecting lateral lobes of typical glandular enlargement.

Urine.—May be infected or contain threads. Moderate residual. A distinction between a fibrous prostate and calculous prostatitis may only be possible after X-ray examination, but pain on micturition is a more prominent feature of the latter.

SUMMARY.

1. An investigation has been undertaken to correlate the structural appearances and clinical features in non-malignant prostatic obstruction.

2. The appearances of 138 autopsy prostates have been studied, and the following histological classification is suggested: (*a*) Glandular enlargement. (*b*) Intermediate form, with some fibrosis in the glandular tissue. (*c*) Fibrous prostates. These three are all examples of benign hypertrophy. (*d*) Calculous prostatitis, in which calculi are associated with chronic inflammatory changes in an atrophic gland.

3. The prostates from 114 consecutive suprapubic prostatectomies (non-malignant) have been examined and classified.

4. The clinical features of these cases have been reviewed and certain differences in the four types noted. The symptoms differ somewhat in each type. Post-operative inflammatory complications are less common in the glandular than the fibrous prostates, and greatest in calculous prostatitis. The mortality of the operation is highest in calculous prostatitis. The end-results are excellent in the glandular type, but less satisfactory in the fibrous and calculous types.

5. Re-appearance of the prostate after operation is the rule in the glandular types, but not in the fibrous.

6. Per-urethral partial prostatectomy is probably the operation of choice for the fibrous prostate, and possibly for calculous prostatitis.

We are indebted to the Honorary Surgeons of Middlesex Hospital for permission to investigate their cases, and to Professors Sir Arthur Keith and James McIntosh for much assistance and advice. One of us (E. G. M.) is working as Bernhard Baron Research Scholar of the Royal College of Surgeons.

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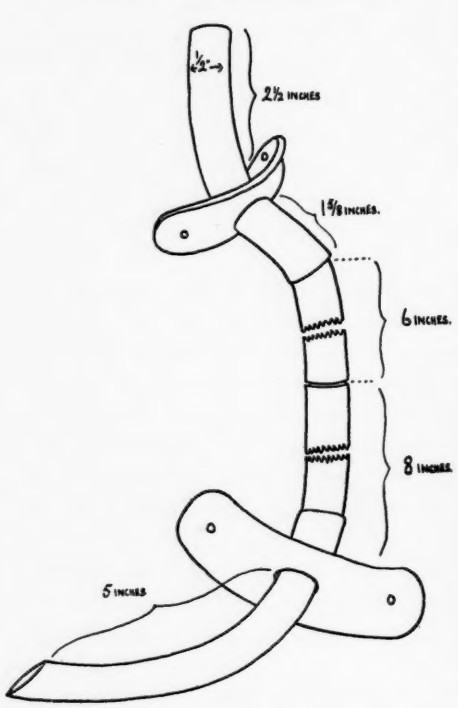
A RUBBER ŒSOPHAGUS.

By ARTHUR EVANS, O.B.E.,
SURGEON, WESTMINSTER HOSPITAL, LONDON.

TWENTY-THREE years ago, for an extensive carcinoma, I removed the larynx, part of the pharynx, the cervical œsophagus, and as much of the thoracic œsophagus as my fingers could reach behind the sternum. This, of course, necessitated a permanent tracheotomy and a permanent gastrostomy. The patient is alive and well to-day.* She takes her food by mouth, masticates and swallows it, and no on-looker would guess there was anything unusual in the process—but an extrathoracic rubber œsophagus has replaced the normal one (*Figs.* 213, 214.)



FIGS. 213, 214.—Tracheotomy tube and rubber œsophagus as worn by the patient.

The patient, a female aged 40, was first seen on Sept. 19, 1909. She complained of hoarseness and increasing difficulty in swallowing solids; this

* This patient was shown and the operation described at the Clinical Section of the Royal Society of Medicine on Nov. 12, 1909; and the patient shown wearing the rubber œsophagus at the Seventeenth International Congress of Medicine, London, 1913.

had been noticed for about five months. Laryngoscopic examination revealed a large malignant mass filling up the lower part of the pharynx, and extending so far forwards that it covered over a large portion of the vocal cords, and almost completely hid the left; one could, however, get a glimpse of the left cord, and it was fixed; the right vocal cord moved freely. By laryngoscopic examination it was not possible to state whether the mass of growth was only postcricoid or whether it extended round the whole circumference of the upper part of the œsophagus. There was no obvious glandular enlargement in the neck. The patient was markedly emaciated.

The malignant nature of the condition was explained to the patient's friends, and also the fact that the complete removal of the growth was considered practically impossible. It was suggested that she should be allowed to remain unoperated upon, and that when the disease had so far advanced that any trouble was experienced in swallowing fluids, gastrostomy should be performed. The risks of an attempt at extirpation were pointed out, and the unenviable condition of the patient, after even a successful operation, was described.

The patient returned on Sept. 25, 1909, and at the urgent request of the patient and friends I consented to attempt the removal of the growth.

Sept. 28, 1909.—Gastrostomy was performed, Abbe's modification of Kader's method being adopted.

Oct. 2, 1909.—An incision was made extending from the hyoid bone to within one inch of the suprasternal notch. The isthmus of the thyroid gland was divided in the middle line. I was anxious to make a close examination of the extent of the growth, and hoped to find I could save some part of the larynx, and maybe, a strip of œsophagus; so I inserted a tracheotomy tube, and then slit up the thyroid cartilage. It was now found that the malignant growth had invaded the whole of the upper orifice of the œsophagus, and a good deal of the pharyngeal wall, and to remove the disease it was necessary to remove the larynx and the upper part of the œsophagus.

The trachea was cut across obliquely, so as to save as much as possible of the posterior wall, a transverse skin incision was made at the suprasternal notch, and the portion of skin between this and the longitudinal incision undermined—through this the trachea was brought and stitched to the skin.

On cutting through the hyoid bone and making a transverse incision immediately below it, a good exposure was obtained of the pharyngeal growth, and it was easy to incise the pharyngeal wall above the growth, and with very little difficulty the lateral walls of the pharynx were freed, and the pharynx with the larynx were separated from above downwards; at the level of the cut trachea it was obvious to the fingers manipulating the œsophagus that the malignant disease extended lower down, so the œsophagus was isolated still lower, and a strong silk ligature passed round it at a level which felt well below the disease; the œsophagus was cut through, and the growth removed (*Figs.* 215, 216).

On slitting up the œsophagus it was evident that the section had been made too near the growth, so the stump was drawn upon by the encircling ligature, the ends of which had been purposely left long, and the œsophagus

was separated for another inch—which was as far as the fingers could reach within the thorax—here the œsophagus was ligatured and cut across.

FIG. 215.—The growth viewed from behind when the pharynx and œsophagus had been opened in the mid-line posteriorly.

The transverse incision, and all of the median—save the lower inch—were now closed with one layer of sutures and the interior packed lightly with gauze. The convalescence was quite uneventful.

Oct. 14, 1909.—The secretions from the mouth, which trickled out of the unclosed part of the wound, came on to the surface immediately above the tracheal opening, and had constantly to be prevented from being sucked into the trachea. I therefore sewed up the lower inch of the opening, and extended it upwards for a corresponding distance.

I had another reason for wishing to alter the position of the pharyngeal sinus to one higher up in the neck, in that I had designed a small celluloid cup, edged with a rubber cushion, which I attempted to keep in close apposition to the opening by means of a rubber band which encircled the neck, and, this cup being connected by a tube with the gastrostomy opening, I hoped it would serve as a means of conducting food and the secretions of the mouth to the stomach.

This arrangement, however, did not work well; in order to prevent leakage it was necessary to fasten the elastic band so tightly round the neck that the patient suffered great discomfort, and even in this new position the

FIG. 216.—Section of growth showing a squamous-celled carcinoma.

pharyngeal opening was too near the tracheal opening, so that any leakage was at once sucked into the trachea, and set up a distressing cough.

Nov. 1, 1909.—I closed the sinus in the lower part of the neck, and made a new one immediately below the hyoid bone; to make this opening permanent, I fashioned four flaps from the surrounding skin, and with them lined the walls of the sinus. Through this opening I passed into the upper pharynx the short end of a rubber empyema tube, using the largest that could be introduced, and this was kept in position by means of a piece of tape passed round the neck and tied into the holes in the flanges of the empyema tube; the long end of the empyema tube was connected with the gastrostomy tube. At the time of this operation I was not able to detect any enlarged glands in the neck.

What remains to be told had best be done by quoting from letters received.

Nov. 24, 1909.—"The swallowing tube does not act well, I am sorry to say, but I hope it will become better as the wound heals."

Dec. 1, 1909.—The sister writes: "All last week the swallowing tube could not act, and we were bitterly disappointed, but since Sunday the scene is changed. Yesterday she had minced pheasant and bread sauce, and vegetables for dinner, then stewed pears and cream, all of which she thoroughly enjoyed. Bread and butter she has, milk, chocolate, and in fact, whatever happens to be going, all by mouth!"

April, 1910.—"The week after Easter I was able to get weighed, and I had gained over a stone since I was in London".

July, 1910.—This patient sent me her wedding card!

Feb. 23, 1911.—"I am glad to tell you I am feeling very fit indeed. If it were not for my cough, which still bothers me, I could say I never felt so well in my life. My whisper seems to be stronger; I do not experience any difficulty in doing my shopping. The swallowing tube acts very well. Some days it is really perfect. When I tell you I went to dinner at the —— Hotel the last time I was in B—— with my husband, and dined in a room with dozens of people, you will agree with me it is very wonderful. Of course, it is not always so well behaved, but I have very little to complain of. I have my cousin staying with me now, and every day we have had long walks of three and four miles—this will tell you I am strong."

March 8, 1912.—"I am sending you a set of tubes to see. The tape passes round my waist, and this keeps the gastrostomy tube in better. I only take this tube out three times a week to boil; it is always quite pure and clean; there never has been any unpleasant smell, or anything disagreeable in connection with it. The throat tube I boil every morning with the tracheotomy tube. I take the latter out at night as well, but only clean it. I find once a day is quite sufficient to boil that. I always wear lace round the throat to breathe through. I have all my dresses cut low in front to suit the tracheotomy tube, and fill in with lace." (*Fig.* 217.)

Oct. 3, 1912.—"I am sending you a snapshot of myself—I thought you would like to see how robust I look (*Fig.* 217). I am feeling so well, and am getting quite fat. I don't know whether you will laugh when I tell you

that I really think there must be some development taking place in the throat, because I discovered last week that I was able to blow out a match—a thing which would have been utterly impossible twelve months ago."

FIG. 217.—Photograph of the patient, showing the style of dress adopted.

April 16, 1913.—"I am feeling very fit indeed. My cough is troublesome sometimes, especially if I take cold, but with that exception I have very little to complain of. My quiet country life suits me, and my people all think I am looking the picture of health. I only have to do my throat twice a day, and the swallowing tube acts very well. The gastrostomy incision is perfectly white and healthy looking. I only take out that tube once a week now, and then it is perfectly clean and there is never anything unpleasant. Everybody at home, thinks my whisper *much* stronger and clearer than when I was home last, and I think it must be, as I never have any difficulty in doing my shopping."

July 9, 1932.—"I am thankful to say I am very well. I have to be careful not to take cold, and if I do a bit too much the tube chafes the entrance to the stomach, and I have to rest a day; but I really lead quite a normal life. We have a large garden and last summer I used up all our fruit by making over 100 lb. of jam—so you see I am not an invalid.

"I often go into B—— for a day's shopping and lunch at a restaurant —so the swallowing tube acts well. No one seems to notice my whisper; and I get no difficulty in being heard, unless it is *very* noisy. Sometimes people say 'What a cold you have', and suggest many remedies, which amuses me greatly.

"I do a bit of Church work too—I am treasurer for our Parish Magazine, a member of the Church Council, District Secretary for the Girl Guides, and do the Altar flowers. I have not had to call in my doctor for three years."

With regard to the rubber œsophagus now being used by the patient, the original empyema tube has been replaced by a curved rubber tube fitted with a flange—its shape is seen in the illustrations (*Figs.* 213, 214). The Jacques catheter originally introduced into the stomach at the time of the gastrostomy, and whose size was No. 9, has been at various times replaced by rubber tubes of larger and larger size, and eventually has attained its present size, a red rubber tubing bore No. 15; this too is fitted with a flange, 5 in. from its lower end, and to this tapes are attached; the tapes being tied around the abdomen keep the gastric tube in position.

OCCLUSION OF THE MAIN ARTERY AND MAIN VEIN OF A LIMB.*

By W. C. WILSON,

ASSISTANT SURGEON, ROYAL HOSPITAL FOR SICK CHILDREN, EDINBURGH, AND DEACONESS HOSPITAL, EDINBURGH.

INTRODUCTION.

Ligation of the main vein of a limb has acquired in recent years a notable popularity among some surgeons, who have employed it as a measure for the relief of certain disturbances of the circulation in the limb. The measure has been applied with particular zeal to conditions of deficient supply of arterial blood, and such application is even accorded the dignity of a surgical principle, usually spoken of as 'restoration of the balance of the circulation'. Furthermore, ligation of the main vein is believed to exert its benefits most fully when an immediate severe deprivation of arterial blood has followed a sudden occlusion of the main artery. Such circumstances, as is well known, are fraught with the gravest possible consequences to the function and life of the limb, and the choice of treatment is one of immediate and vital importance. The question of the application of venous ligation is obviously, therefore, not trivial. The present work is an experimental study of the effects of venous ligation in sudden arterial occlusion.

The origin of the practice of deliberate venous ligation in recent times has been so thoroughly traced by Brooks[5] as to make a detailed historical review unnecessary here. Ligation of the main vein of a limb probably owes its present standing as a therapeutic procedure very largely to the undoubted success which attended its use when the main artery had been ligated for the cure of an arteriovenous communication in the main vessels. The condition of arteriovenous communication is not considered in this paper. Venous ligation has also been employed in conditions of chronic arterial disease.[10, 11, 15, 16] Reports of the results are conflicting, and the benefits seem to be temporary and uncertain.

There is, however, a widespread and hitherto unquestioned belief that, if the main artery of a limb be suddenly occluded, as, for example, by necessary ligation, then ligation of the main accompanying vein lessens the chances of subsequent development of gangrene. Certain clinical and experimental observations have been cited to justify the belief. The most valuable and suggestive evidence on the clinical side is furnished by the personal experience of Sir George Makins[12] in the treatment of wounds of large vessels

* This work was carried out at the Department of Surgery, University of Chicago, during tenure of a Rockefeller Foundation Fellowship in Surgery.

during the war. The main experimental support is the work of Brooks and Martin,[3] and an extension of their experiments by Holman and Edwards,[7] who claimed that ligation of the vein at a level higher than arterial ligation still further reduced the incidence of gangrene.

The favourable results of simultaneous venous ligation seem to have been universally accepted. The mechanism whereby gangrene is diminished is still, however, disputed. Many theories have been submitted in explanation, together with experimental results frequently conflicting and contradictory. A brief summary of these views and investigations is inserted at this point.

All workers are agreed that after occlusion of the main artery occlusion of the main vein causes a rise in pressure in the arteries distal to the obstruction. It was found that the pressure rose also in the veins and to a greater extent than in the arteries.[3] All recorded experiments on ligation of the vein at a level higher than the arterial occlusion (proximal venous ligation) show that the higher the level at which the vein is ligated, the greater is the rise in limb arterial pressure.[7, 17] The temperature of the tissues of the distal part of the limb falls after arterial occlusion, and the fall is accentuated by subsequent occlusion of the vein.[3]

Experiments which were quoted as indicating that the volume flow of blood per minute through the limb was increased by ligation of the vein were destructively criticized by Brooks. By direct methods the approximate total volume of blood flowing through the whole limb in unit time has been measured by Montgomery,[14] who found that proximal venous ligation caused a considerable reduction of volume flow. His experiments have been repeated and amplified in this investigation, and their significance is discussed later. Injection of the vessels at intervals after vascular occlusion shows that the calibre of the distal arteries is increased for some time by venous ligation.[17, 18, 19] There is some discrepancy in the estimates of the time during which the calibre remains increased.

Four main theories have been propounded to explain the beneficial effects of venous ligation :—

1. The theory that ligation of the vein 'restores the balance of the circulation'. This implies a belief that, in occlusion of the artery only, the capacious main vein affords a too ready outlet for the diminished quantity of blood reaching the part below the occlusion. The blood tends to supply mainly the proximal tissues of this part and is not dispersed to the distal tissues. There is thus a condition of imbalance of inflow and outflow, which, it is claimed, can be corrected by venous occlusion.

A closely allied idea is that venous ligation keeps the blood in longer contact with the tissues, which are, presumably, enabled thus to extract more oxygen from it.

2. The theory, formulated by Brooks, of a more homogeneous distribution of the arterial blood. The main favourable influence, according to him, is the raised intravascular pressure, which serves to keep the capillaries patent in areas in which they would otherwise collapse, and thus to distribute the arterial blood more uniformly throughout the tissues.

3. The theory of the development of a 'richer collateral bed' is the

consequence of injection experiments. The exact significance of the increased size of the vessels is not clearly stated.

4. The theory of a 'masked sympathectomy' is advanced by Leriche and Fontaine.[9] They believe that the initial rise of limb arterial pressure after venous ligation is associated with a transient peripheral vasoconstriction, which is succeeded in some minutes by an active vasodilatation, and that these effects are produced by reflex action along sympathetic nerve fibres.

Our experiments began with a study of the effects of temporary venous occlusion on the incidence of gangrene caused by arterial obstruction. Evidence of beneficial results from permanent or temporary occlusion of the main vein, however, was not forthcoming. A comprehensive re-investigation of the subject seemed desirable and will now be reported.

SURVIVAL EXPERIMENTS ON LIGATION OF ARTERIES AND VEINS.

DESCRIPTION OF EXPERIMENTS.

These experiments were carried out on rabbits. The total number for consideration is 109, divided into seven series :—

Series 1.—Ligation of the external iliac artery : 10 animals.

Series 2.—Ligation of the common iliac artery : 10 animals.

Series 3.—Ligation of the common and external iliac arteries (both arteries) : 30 animals.

Series 4.—Ligation of both arteries and of the common iliac vein : 30 animals.

Series 5.—Ligation of both arteries and of the inferior vena cava : 10 animals.

Series 6.—Ligation of both arteries and temporary ligation for twenty-four hours of the common iliac vein : 11 animals.

Series 7.—Ligation of both arteries and temporary ligation for twenty-four hours of the inferior vena cava : 8 animals.

Series 1 afforded a study of the effects of mild ischæmia. *Series* 2 and 5 were a test of the results of Holman and Edwards, while *Series* 3 and 4 formed a repetition of the work of Brooks and Martin but in a larger series of experiments. *Series* 6 and 7 were an investigation of temporary venous obstruction. A common assumption which holds that the beneficial effects of venous occlusion are exercised during the first twenty-four hours is thus subjected to experimental trial.

The vessels of the right posterior extremity were exposed by a mid-line abdominal incision. Arteries were divided between ligatures, veins were ligated. Temporary venous ligation was carried out in the following way. A piece of vaselined braided silk was passed through muscle on each side of and below the vein, and tied in a loop ligature. The vessel wall was thus protected by muscle from direct pressure of the ligature. The long end of the loop was brought out through a stab-wound in the flank and the ligature was released and removed by traction on the end at the required time. All cases in which constriction of the vein from fibrosis was found subsequently are omitted from consideration. The animals were kept alive for periods as long as 230 days.

EFFECT OF VASCULAR LIGATIONS ON FUNCTION AND STRUCTURE OF THE LIMB.

The animals have been divided into four groups as follows: *Group I*—Those which developed gangrene. *Group II*—Those with severe circulatory disturbances, but without gangrene. *Group III*—Those with moderate circulatory disturbances, without gangrene. *Group IV*—Those with slight and apparently temporary circulatory disturbances, without gangrene. Since the changes for any one group were almost identical in all series, each group is discussed without special reference to the vascular ligation. The description of the changes is necessarily brief.

Group I.—In this group there was death of the skin or of the skin and deeper structures together. Gangrene was of three main types: *Type A*—Massive gangrene, involving all the tissues of a portion of the limb, such as the whole foot or leg below the knee. *Type B*—Late gangrene of peripheral parts, such as the digits or portions of digits. *Type C*—Very late and limited gangrene of the peripheral parts. One case of gas gangrene occurred; all others were cases of dry gangrene.

Type A.—The features of dry gangrene became apparent between the second and seventh days. From the commencement the whole limb was powerless, cold, and of diminished volume, and the tendon reflexes were absent. Swelling appeared after a period of between sixteen and forty-eight hours and was limited to the portion ultimately surviving. It was more marked after venous ligation and especially after ligation of the inferior vena cava. The muscles which ultimately died *en masse* responded to direct stimuli on occasion for as long as eight hours after ligation. Sometimes they passed into a condition resembling rigor mortis between four and ten hours.

At post-mortem interpretation of gangrene was not always easy during the first forty-eight hours, and all cases of doubtful gangrene are included in *Group II*. In the surviving portion there were œdema, subcutaneous and intermuscular, congestion of superficial vessels, and, in the muscles, diffuse changes which became distinctive only after forty-eight hours. Most muscles had undergone necrosis which involved the whole or part of the muscle. The appearance was that of an anæmic infarct, the pale yellow necrotic area being separated from living muscle by a mottled reddish demarcation zone. Necrotic muscle fibres were swollen, disintegrated, and structureless, separated by fluid and cellular débris, and the nuclei did not stain. The demarcation zone was characterized by dilated small vessels and by a proliferation of leucocytes among degenerated fibres. Some of the fibres had a deep basic stain, after the third day, from deposition of calcium.

Type B.—In the early stage the changes during life were similar to those in *Type A*. Swelling was generalized but most marked in certain areas. Those were found later to contain necrotic muscle. Later, during the second week, as swelling subsided, such areas became indurated. Gradual return of voluntary power began during the second week, but was masked later by the development of *contractures*, which from the third week onwards steadily produced fixation at the joints, and finally converted the limb into a rigid

deformed appendage (*Fig.* 218). Gangrene never appeared before the fourth week. It affected the digits only, and was preceded by changes in the skin, such as loss of hair, discoloration, and ulceration. Gangrene, once developed, frequently spread proximally.

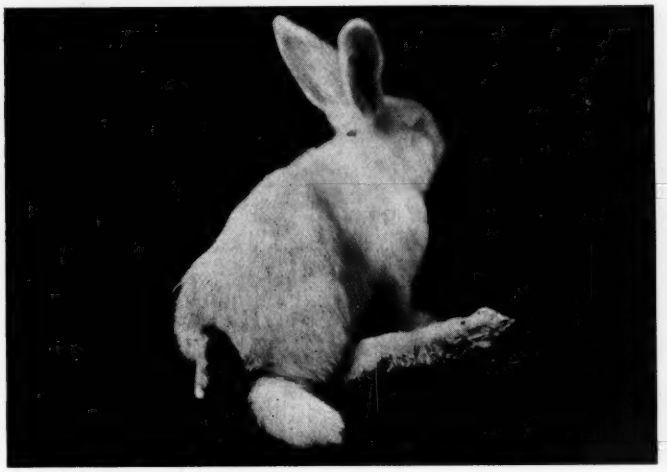


FIG. 218.—Contractures of right hind limb 65 days after ligation of both arteries. Gangrene of digits developed on 70th day.

At post-mortem it was found that in the surviving portion of the limb many muscles had become necrotic in whole or in part. The necrotic areas

FIG. 219.—Microphotograph showing changes at 28th day after arterial ligation. Note part of a necrotic zone surrounded by demarcation zone of young fibrous tissue and atrophied fibres. (× 30.)

were surrounded and replaced by fibrous tissue (*Fig.* 219), which caused shortening and fixation of muscles and thus contractures. Surviving muscle was thin and atrophied and often infiltrated to a slight extent by young

fibrous tissue. Replacement of necrotic muscle was a very slow process. No rapidly developing massive fibrosis of muscle was noted. The degree of fibrosis and the incidence and development of contractures were not materially affected by venous ligation.

Type C.—During life the main feature was marked muscle weakness for as long as four weeks, followed by a slow and finally almost complete recovery of power. Very little swelling, no induration, and no contractures were noted. The limb was very thin. Gangrene appeared in the digits after the skin changes noted above, but remained strictly limited. After spontaneous amputation, the stump healed.

The animals were killed after several months. The muscles of the limb were thin. Necrosis had occurred in only a few muscles and there were corresponding replacement changes. The other muscles showed the changes of atrophy.

Group II.—The majority of animals in this group died before the twelfth day. During life the changes were very similar to those in *Type B* of gangrene at the corresponding time. They might be classified as cases of potential gangrene, and probably most, had they survived, would have developed late peripheral gangrene. Those surviving beyond the second week developed contractures.

In this group are included also all cases of doubtful early gangrene. There were six in *Series* 3 and five in *Series* 4.

At post-mortem the alterations were similar to but less diffuse than those in the surviving portion of the limb in *Type A* gangrene, and probably they represent an early stage of the changes found later in *Type B* gangrene. The distribution of muscle necrosis was very similar to that in the latter. In the late stage the appearances, apart from gangrene, were identical with those in *Type B* gangrene.

Group III.—All animals in this group died at an early stage before recovery of power was complete. The probable outcome, so far as could be judged, was a full functional recovery or a late limited gangrene as in *Type C* gangrene. The structural changes were those of scattered necrosis of muscle.

Group IV.—The ultimate result in this group was an apparently complete recovery of function in the limb after a period of muscle weakness varying from three days to eleven weeks. In some the limb became thin and atrophied to a degree corresponding roughly to the duration of muscle weakness. In the early stages swelling was slight and transient, or absent, and subsequently no induration or contractures were noted.

In *Series* 1 recovery occurred in all within a few days. In *Series* 2 weakness was more prolonged, lasting on an average for eighteen days. In the other series the average duration of weakness was as follows: in *Series* 3, twenty-four days; in *Series* 4, thirty-eight days; and in *Series* 5, forty-seven days.

At post-mortem the changes were atrophy of muscle, and, in some animals, necrosis of muscle affecting usually one or more muscles of the quadriceps group.

DISTRIBUTION OF MUSCLE NECROSIS.

Involvement of the different groups of muscles in cases with scattered muscle necrosis is shown in *Table I.* Cases of very diffuse necrosis are not included.

Table I.—Involvement of Muscle Groups in Cases of Scattered Necrosis of Muscle.

Series	Ligation	Total Cases	Quadriceps	Posterior Thigh Group	Calf Group	Leg Extensors
1	External iliac artery	1	1	—	—	—
2	Common iliac artery	7	6	1	—	—
3	Both arteries	10	10	6	8	4
4	Both arteries and common iliac vein	13	12	6	9	8
5	Both arteries and inferior vena cava	4	3	3	3	3
6	Both arteries and temporary ligation for 24 hours of common iliac vein	3	3	2	2	1
7	Both arteries and temporary ligation for 24 hours of common iliac vein ..	2	1	1	2	1
	Totals	40	36	19	24	17

The distribution of necrosis was remarkably regular both as regards groups of muscles and individual muscles of each group. For example, the quadriceps group was almost invariably affected and the superficial calf group with great frequency. The distribution of necrosis indicates the distribution of the blood entering the limb. The regularity of distribution of necrosis is evidence that the distribution of blood entering the limb after arterial occlusion is determined by the anatomical situation of the conducting collateral channels. Obviously, moreover, the distribution of blood is altered by arterial occlusion but the proximal muscles are not supplied at the expense of the distal. Necrosis of muscle is a process of infarction of a considerable portion of the muscle and not the focal necrosis which would be expected if, as Brooks thought, the capillaries in certain areas were closed while those in the immediate neighbourhood remained open. Ligation of the vein does not affect the distribution or extent of muscle necrosis.

RESULTS.

The distribution of each series into groups is shown in *Table II.*

The incidence of gangrene is equal in *Series* 3 and 4, and is not appreciably altered if one includes as gangrene those doubtful cases in *Group II* previously mentioned. It is not conspicuously different in *Series* 6 and 7, where temporary venous occlusion was tried. It is *increased* in *Series* 5 by

ligation of the inferior vena cava. The division of the cases of gangrene into the different types is shown in *Table II*. The distribution into types is notably similar in *Series* 3 and 4, while in *Series* 5 the proportion of massive gangrene is higher. In *Type A* accurate determination of the time of appearance of gangrene was not possible, though it can be stated that there was no appreciable difference in the different series. In *Type B* the earliest date of appearance of gangrene was the twenty-first day and the latest the seventieth day. The average time of appearance of this type of gangrene was

Table II.—Results of Vascular Ligations.

Series	Ligation	Total Cases	Group I	Group II	Group III	Group IV	Death before 10th day	Types of Gangrene		
			Gangrene	Severe Disturbances	Moderate Disturbance	Slight Disturbances		*A*	*B*	*C*
1	External iliac artery ..	10	0	0	0	10 = 100%	0	—	—	—
2	Common iliac artery ..	10	1 = 10%	0	0	9 = 90%	1 = 10%	1	—	—
3	External and common iliac arteries ..	30	13 = 43%	8 = 27%	2 = 7%	7 = 23%	11 = 37%	4	9	—
4	Both arteries and common iliac vein ..	30	13 = 43%	10 = 34%	3 = 10%	4 = 13%	8 = 27%	3	8	2
5	Both arteries and inferior vena cava..	10	6 = 60%	2 = 20%	0	2 = 20%	3 = 30%	4	1	1
6	Both arteries and temporary ligation of common iliac vein for 24 hours ..	11	5 = 45%	2 = 18%	0	4 = 37%	1 = 9 %	2	3	—
7	Both arteries and temporary ligation of inferior vena cava for 24 hours ..	8	4 = 50%	0	1 = 12%	3 = 38%	3 = 30%	2	2	—

identical in *Series* 3 and 4, namely the thirty-fifth day. In *Type C* gangrene appeared in one case during the sixth week and in the others during the eleventh week.

The incidence of full recovery is shown in *Table II* by the figures for *Group IV*. The difference between *Series* 3 and 4 is perhaps scarcely decisive, but the average duration of muscle weakness in the different series suggests strongly that ligation of the vein delays the return of power. Ligation of the inferior vena cava delays functional recovery still further.

The mortality during the first ten days after ligation is shown in *Table II.* The figures provide evidence of a possible benefit from venous ligation, since the mortality is higher after arterial ligation alone. Similar but more striking results were obtained by Brooks and Martin. The cause of death after ligation of large arteries will not be discussed in this paper.

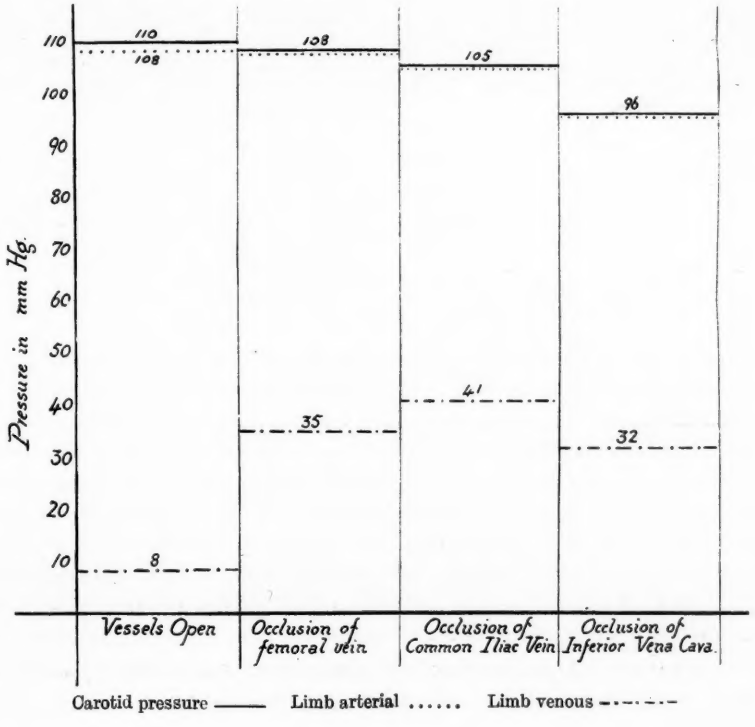
THE IMMEDIATE RESULTS OF OCCLUSION OF THE MAIN VESSELS OF A LIMB.

The immediate effects which have been studied are those related to the intravascular pressure, the mass of blood in the limb, the volume flow of blood per minute, and the oxygen consumption of the tissues of the limb per minute. For these experiments dogs were used. The animals were anæsthetized by intravenous injection of sodium barbital, 250 mgrm. per kilo. of body weight. In most instances heparin was injected to prevent clotting of blood. The amount was 30 mgrm. per kilo. of body weight.

INTRAVASCULAR PRESSURE.

The vessels occluded were those of one hind limb. Continuous records were taken of pressure in the carotid artery and of the limb arterial and limb venous pressures below the level of occlusion. The limb pressures were recorded from cannulas tied into a side branch of each main vessel or from

Table III.—Results of Venous Occlusion without Arterial Occlusion.



T-cannulas in the main vessels. Figures for arterial pressure signify mean arterial pressure in mm. Hg.

1. **Venous Occlusion without Arterial Occlusion.** — The femoral vein, iliac vein, and the inferior vena cava were occluded in turn. The average results of six experiments are shown in *Table III.*

Points.—

a. The limb arterial pressure follows closely the carotid pressure.

b. Occlusion of the femoral vein is without effect on the limb arterial pressure. Occlusion of the iliac vein or of the inferior vena cava causes a fall in pressure in the carotid and limb arteries. The fall is due to a lessened return of blood to the heart.

2. **Arterial Occlusion.**—The changes in the limb pressures and the carotid pressure after occlusion of the main artery were measured. The results of fifteen experiments are shown in *Table IV.* The figures refer to mean pressures in mm. Hg. at ten minutes after occlusion.

Table IV.—Changes in Limb Pressure and Carotid Pressure after Arterial and Venous Occlusion.

	Femoral Vessels						*Iliac Vessels*					
Experiment No.	Carotid Pressure	Arterial Occlusion		Rise after Venous Occlusion		Experiment No.	Carotid Pressure	Arterial Occlusion		Rise after Venous Occlusion	
		L.A.P.	L.V.P.	L.A.P.	L.V.P.			L.A.P.	L.V.P.	L.A.P.	L.V.P.
10	125	62	—	16	—	6	112	58	—	3	—
24	90	48	—	7	—	8	120	84	—	0	—
25	110	78	4	2	17	13	136	53	—	7	—
26	110	64	4	0	20	16	112	16	4	28	30
33	150	106	6	9	26	19	120	36	5	5	10
34	144	78	4	16	18	20	122	76	5	0	4
35	122	78	4	0	6	38	130	57	6	19	24
40	90	42	4	0	3						
Average	118	69	4·2	6·2	15	Average	126	54	5	9	17

L.A.P. = Limb arterial pressure in mm. Hg. L.V.P. = Limb venous pressure in mm. Hg.

Points.—

a. The effect on the mean carotid pressure is insignificant.

b. The limb arterial pressure falls sharply, and then, recovering quickly, assumes a level (*Fig.* 220). If a high level is reached, pulsations return. The level varies greatly in different animals.

3. **Occlusion of the Accompanying Main Vein after Occlusion of the Main Artery.**—The results of fifteen experiments are shown in *Table IV.*

Points.—

a. In 50 per cent of experiments the effect of venous occlusion on the limb arterial pressure is negligible. In the remainder a rise of pressure occurred. The rise is therefore inconstant.

b. The rise in venous pressure is always greater than any simultaneous rise in pressure in the arteries. The same observation was made by Brooks and Martin.

c. Occlusion of the main vein has a local action, whereby it raises the pressure in the veins and sometimes in the arteries of the limb, and a more general action, whereby it lessens the return of blood to the heart and may thus cause a fall in the general systemic pressure.

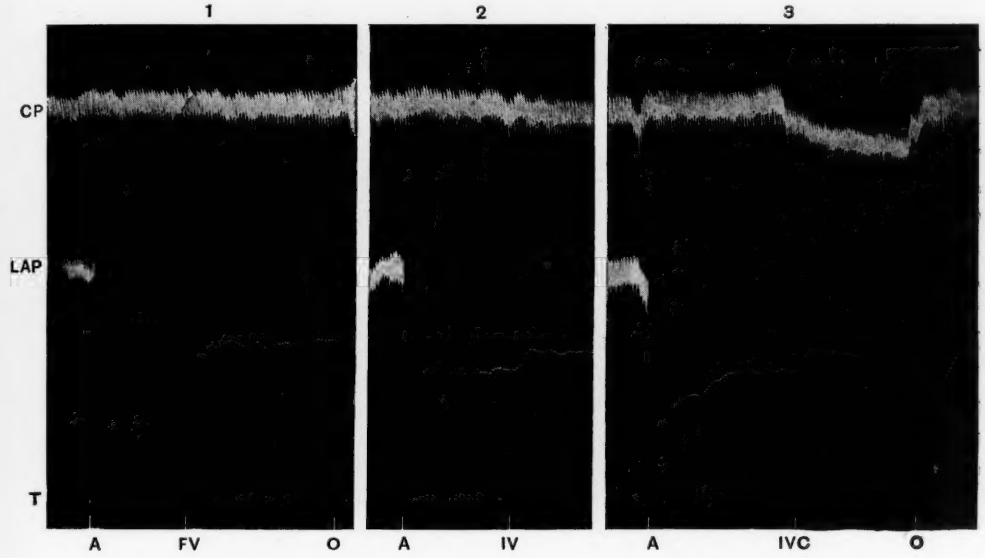


FIG. 220.—Experiment 46. Successive occlusion of femoral artery and main veins at different levels. (1) Occlusion of femoral vein at FV; (2) Occlusion of iliac vein at IV; (3) Occlusion of inferior vena cava at IVC. At A, occlusion of femoral artery; at O, release of vein. Note decrease of rise from 1 to 3. CP, Carotid pressure; LAP, Femoral artery pressure; T, Time marker in 5 seconds.

4. **Occlusion of the Vein at a Level Higher than that of Arterial Occlusion.**—The effect of such occlusion was tested in six experiments. In three experiments the results were the reverse of those previously reported (*Fig.* 220). In the others the increased rise was found. The phenomenon is therefore not constant.

An important fact was noted. On two occasions occlusion of the inferior vena cava caused a marked fall in general arterial pressure, during which the limb arterial pressure fell to below the level it had attained after arterial occlusion alone. In such instances the effect of occluding the vein on the general systemic pressure outweighs the local effect on the intravascular pressure in the limb.

5. **Mechanism of Rise of Pressure.**—The theory of Leriche and Fontaine was tested with the following results:—

a. The rise of arterial limb pressure, if present initially, occurs after

denervation of the limb and removal of the entire lumbar sympathetic chain.

b. No change in the volume of a limb occurs during occlusion of the main vessels of the opposite limb, if the carotid pressure is unaffected.

c. During occlusion of the main artery no rise of limb arterial pressure occurs on occlusion of the contralateral main vein.

It may be concluded that the rise of pressure is independent of the integrity of the nerves of the limb. There was no evidence of a sympathetic reflex action involving the vessels either of the same or of the opposite limb.

The reaction of the vessels of the limb to occlusion of the main artery and vein must also be considered.

In order to determine the influence on the tone of the vessels of occlusion of the main vein with occlusion of the main artery, the following experiment was devised. The main artery and vein and the larger nerves of the limb were isolated in the upper part of the thigh. All other structures were severed, including the bone, so that blood entered and left the limb only by the main vessels. The limb pressures were recorded. On occlusion of the artery the pressure in arteries and veins reached a level of about 5 mm. Hg. in three to four minutes. After simultaneous occlusion of artery and vein the same result was obtained in about five to six minutes, the venous pressure rising for a brief period as the arterial pressure began to reach a level. Division of the nerves did not affect the result.

Thus occlusion of the vein does not prevent the rapid loss of vascular tone which follows complete arterial occlusion. That its effect on vascular tone is different in lesser degrees of arterial obstruction is certainly unlikely. More probably it accentuates the active vasodilatation on the arterial side. A similar experiment on the vascular tone in man has been described by Kendrew.[8]

6. **Duration of the Rise of Limb Pressure.**— In four experiments the artery and vein of one limb and the artery of the opposite limb were divided between ligatures. The pressures in artery and vein of each limb were measured after twelve hours in two experiments and after twenty-four hours in two. It was found that the rise of pressure in the limb arteries after venous occlusion did not last for more than twelve hours.

MASS OF BLOOD IN THE LIMB.

Plethysmographic tracings showed that the volume of the limb fell after arterial occlusion and then, on subsequent venous occlusion, rose to a level well above the normal. The increased volume affected the most distal parts, as shown by a record of foot volume, and its duration was variable. The same changes occurred after denervation but were less marked, since the vessels were initially relaxed.

The quantity of blood in the limb is, therefore, greater than normal for a variable period after occlusion of both vessels. It follows that there must be considerable increased filling of vessels, most probably on the venous side, and a marked slowing of the flow of blood.

VOLUME FLOW OF BLOOD PER MINUTE, OXYGEN UTILIZATION, AND OXYGEN CONSUMPTION OF THE TISSUES PER MINUTE.

The experiments in this section were carried out in conjunction with Dr. M. L. Montgomery. The *Stromuhr* designed and described by him was used for the measurement of volume flow. A brief consideration of his experiments, already quoted, is required here, and the experimental conditions must be clearly understood. The flow which was measured includes the greater part of the blood going to the posterior extremity. The blood enters the branches of the main vessel above the obstruction on the femoral artery. A part passes through the capillary bed of the tissues which these branches normally supply. The remainder enters the limb below the obstruction through collateral channels. The tissues of the whole limb may be divided into the part above the obstruction and the part below, i.e., into 'unobstructed' and 'obstructed' portions of the limb. The effect of proximal venous ligation at least is to diminish the total flow through the whole limb.

We are concerned, however, mainly with the flow through the obstructed portion, and, unfortunately, there is as yet no accurate method of measuring it. Nevertheless, there is much indirect evidence of a decreased flow through the obstructed portion after venous ligation, and it may be assumed that venous ligation which, as Montgomery showed, causes a diminution of the total volume flow, produces also a corresponding decrease in the flow through the obstructed portion.

If one accepts the hypothesis that the volume flow through the obstructed portion varies directly with the total volume flow, one can obtain an estimate of the oxygen consumption of the tissues of the obstructed portion per minute. To do so the total volume flow is measured and also the oxygen utilization (i.e., the difference in oxygen content of the arterial and venous blood) below the level of obstruction. The product of volume flow per minute and oxygen utilization gives an index of the oxygen consumption of tissues per minute.

Oxygen Utilization. — The following technique was adopted. From T-cannulas in iliac and femoral vessels samples of blood were withdrawn under oil before occlusion, during occlusion of the femoral artery, and during occlusion of artery and vein. The oxygen content of each sample was measured in a Van-Slyke-Neil apparatus. Blood from the iliac vein may be considered as blood from the tissues of the whole limb, that from the femoral vein as from the tissues of the obstructed part. A typical example is shown in *Table V*.

Results.—

a. The arterial oxygen content remained constant above and below the obstruction. Therefore the collateral channels are larger than capillaries.

b. The oxygen utilization and the co-efficient of utilization rose in both iliac and femoral blood after arterial occlusion. The rise was higher below the occlusion.

c. On subsequent venous occlusion there was a further rise in utilization and co-efficient of utilization, and the rise was more marked below the obstruction.

These results indicate an increased stasis after venous occlusion.

Table V.—Oxygen Utilization.

	Iliac Artery O_2 Content	Iliac Vein O_2 Content	Iliac Vessels O_2 Utilization	Iliac Vessels Coeff. of Utilization	Femoral Artery O_2 Content	Femoral Vein O_2 Content	Femoral Vessels O_2 Utilization	Femoral Vessels Coeff. of Utilization
	Vols. %	Vols. %	Vols. %		Vols. %	Vols. %	Vols. %	
1 Vessels unoccluded	20·04	15·45	4·59	0·23	20·04	12·63	7·41	0·37
2 Femoral artery occluded ..	20·04	13·01	7·03	0·35	20·04	9·91	10·13	0·51
3 Femoral artery and vein occluded ..	20·10	10·95	9·15	0·46	20·10	7·84	12·26	0·61

Oxygen Consumption of the Tissues per Minute.—

a. The Obstructed Portion.—The oxygen consumption of the tissues of the obstructed portion of the limb was estimated as the product of the volume flow through the iliac artery and of the oxygen utilization of blood in the femoral vessels. The consumption was measured with the vessels unoccluded, the femoral artery occluded, and the femoral artery and vein occluded. The occlusion was repeated while recording the pressures in the femoral vessels. *Tables VI* and *VII* are typical examples, and a record is shown in *Fig.* 221.

Table VI.—Volume Flow and Limb Pressures.

Vessels	Carotid Pressure in mm. Hg.	L.A.P.	L.V.P.	Vol. Flow per Min.
				c.c.
No occlusion	138	128	6	95·60
Occlusion femoral vein ..	139	129	86	64·40
Occlusion femoral artery ..	144	78	3·8	30·46
Occlusion femoral artery and vein	144	94	22	25·81
No occlusion	142	128	4·5	87·26

L.A.P. = Limb arterial pressure in mm. Hg. L.V.P. = Limb venous pressure in mm. Hg.

Table VII.—Estimated Oxygen Consumption of Limb.

Vessels	Carotid Pressure in mm. Hg.	Vol. Flow per Min.	A—V.O_2 Diff. in Femoral Vessels	Coeff. of Utilization	Estimated O_2 Consumption per Min.
		c.c.	Vols. %		c.c.
No occlusion	108	74·03	8·50	0·38	6·20
Occlusion femoral artery	100	28·60	12·64	0·57	3·62
Occlusion femoral artery and vein ..	98	22·40	15·26	0·68	3·42

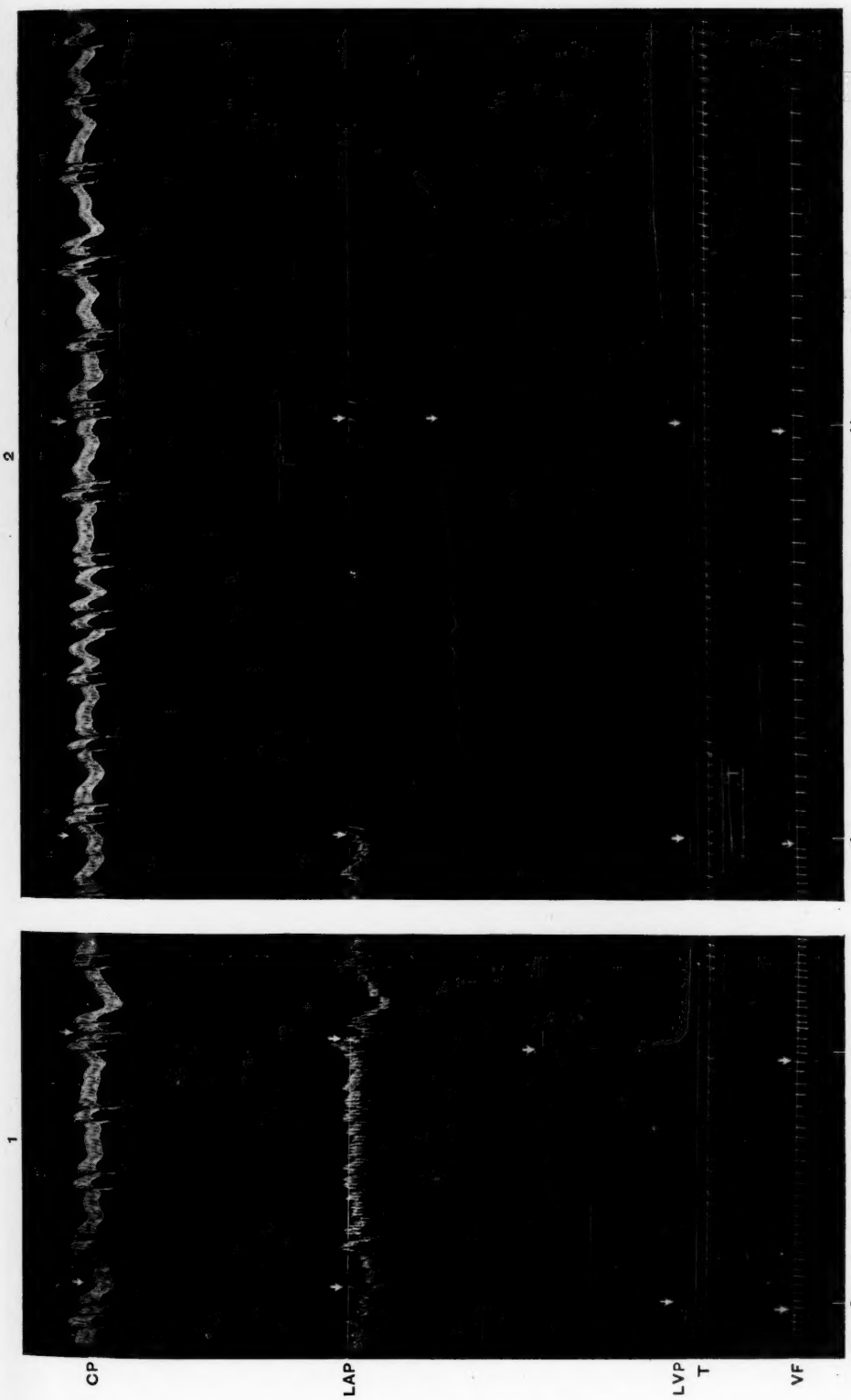


FIG. 221.—Experiment 34. Successive occlusion of femoral vessels. Effect on volume flow through iliac artery per minute, carotid and limb pressures. C—O, Occlusion of femoral vein alone: flow 64·40 c.c. per min. A—V, Occlusion of femoral artery alone: flow 30·46 c.c. per min. After V, Occlusion of femoral artery and vein: flow 25·81 c.c. per min. CP, Carotid pressure; LAP, Femoral artery pressure; LVP, Femoral vein pressure; VF, Record of volume flow; T, Time-marker in 5 seconds.

The volume flow falls after occlusion of the artery and still further after subsequent occlusion of the vein. The oxygen utilization rises, but the oxygen consumption per minute as calculated by this method decreases after venous occlusion.

b. The Whole Limb.—The oxygen consumption was measured as the product of volume flow through the iliac artery and of the oxygen utilization in the iliac vessels. The results were similar to those in the previous experiments.

SUMMARY OF IMMEDIATE EFFECTS OF OCCLUDING THE MAIN ARTERY AND MAIN VEIN OF A LIMB.

1. *After Occlusion of the Main Artery of a Limb.—*

a. The limb pressures, arterial and venous, fall.

b. The mass of blood in the limb and the volume flow of blood per minute are diminished.

c. The oxygen utilization, and co-efficient of utilization, of blood in the main vessels above and below the occlusion are increased.

d. The oxygen consumption per minute of the tissues of the whole limb and of the tissues below the obstruction is diminished.

2. *On Subsequent Occlusion of the Main Vein at the Same Level.—*

a. The limb pressures may increase, but the rise in limb arterial pressure is inconstant and frequently insignificant, and probably in any case lasts for no more than twelve hours. The rise of arterial pressure does not depend upon the integrity of the nerves to the limb or of the lumbar sympathetic system. The rise is probably purely a mechanical effect of obstruction to the venous outflow. There is no evidence of increased tone in the vessels. The rise in limb venous pressure is always greater than the corresponding rise in limb arterial pressure.

b. The mass of blood in the limb becomes greater than normal.

c. The volume flow of blood per minute is further diminished through the whole limb, and probably through the tissues below the obstruction.

d. The oxygen utilization and coefficient of utilization of the blood in the main vessels above and below the occlusion are further increased.

e. In spite of increased oxygen utilization, the oxygen consumption per minute of the tissues of the whole limb and of the part below the occlusion is still further diminished.

3. *Occlusion of the Vein at a Higher Level as Compared with Occlusion at the same Level as Arterial Occlusion.—*

a. The limb arterial pressure may rise still higher. The rise is inconstant and the result may be a fall associated with a dangerous fall in general arterial pressure when the lower end of the inferior vena cava is occluded.

b. The reduction in volume flow per minute is more marked and always considerable.

DISCUSSION.

The results of the survival experiments indicate definitely that ligation of the main vein does not lessen the incidence of gangrene which follows ligation of the main artery of a limb. Venous ligation does not materially

affect the nature, extent, or time of appearance of subsequent gangrene, and probably lessens the chances of complete recovery of function. It delays the return of power to the muscles of the limb. The only apparently beneficial effect of obstructing the veins was a reduction in the immediate mortality following occlusion of large arterial channels. Temporary occlusion of the main vein for twenty-four hours confers no certain benefit.

The main experimental basis for the belief that ligation of the vein decreases the incidence of gangrene is the work of Brooks and Martin. This investigation failed to confirm their work in a larger series of experiments, in which the animals have been kept alive for longer periods, and in which special effort has been made to exclude fallacies in technical execution of the operations, and in interpretation of gangrene.

The claim put forward by Holman and Edwards, that ligation of the vein at a higher level still further diminishes the incidence of gangrene, is based on experiments which are open to objection on two points. Firstly, they employed no control series of ligation of artery alone, but used the figures quoted by Brooks and Martin for comparison with their results. Secondly, they did not in all experiments employ the arterial ligation of Brooks and Martin. In 18 experiments they reported gangrene in 11 per cent of cases when the common iliac artery and inferior vena cava were ligated. In the present *Series* 2 ligation of the common iliac artery alone gave gangrene in only 10 per cent of cases. The results in the present *Series* 5, which forms a repetition of the remainder of their experiments, are completely at variance with theirs.

In view of the results of the survival experiments it might seem unnecessary to review the theories of the supposed beneficial action. These theories, however, have become to some extent current belief, and are based on experimental results which are of considerable theoretical importance.

The theory of 'restoration of the balance of the circulation' is an example of vague phraseology. There occurs after venous occlusion a further diminution of the volume flow of blood per minute and of the rate of flow. Such conditions can scarcely be designated a restoration of circulatory balance.

Venous ligation does not alter the incidence of gangrene or the severity or distribution of muscle necrosis. Therefore it does not effect a dispersion of the arterial blood to the distal parts. For the same reasons Brooks' theory of a more homogeneous distribution of arterial blood is not acceptable.

The belief that ligation of the vein aids by keeping the blood in longer contact with the tissues cannot at present be countenanced. The oxygen consumption per minute of the tissues of the limb as a whole, and of the obstructed portion, is lessened by venous ligation, although the limb contains a quantity of blood greater than normal and the oxygen utilization of the blood is increased. Volume flow per minute is apparently the most important factor for oxygen consumption.

The observation that for some time after venous ligation the calibre of the arterial vessels is increased has given rise to the theory of a 'richer collateral bed'. The observation is very probably correct, but has no great bearing on the question of nutrition of tissues or gangrene.

It is not agreed that venous occlusion acts as a 'masked sympathectomy'. The initial rise of limb arterial pressure may be only temporary, and an active dilatation of vessels on the arterial side is possible, and even probable. The reactions of the vessels to occlusion, however, are independent of the integrity of the nerves to the limb.

The dangers of marked venous obstruction in a limb are well known. In clinical cases gangrene has been reported from venous obstruction without arterial obstruction.[1, 6] Thus temporary occlusion would seem preferable to permanent occlusion of the vein. Moreover, it is usually stated that the supposed benefits accrue during an early period. However, no beneficial action of temporary venous occlusion for twenty-four hours has been found in this work.

The experiments on proximal venous ligation have demonstrated that this procedure has effects which are decidedly deleterious.

Special Features.—Two features merit further consideration—namely, the special vulnerability of tissues to ischæmia, and the occurrence of contractures.

Experiments by Brooks[2] showed that with a permanent severe deprivation of arterial blood the skin of a limb was more extensively affected than the muscles, while, if the deprivation were temporary, the changes were more severe and extensive in the muscles. He concluded that skin is less tolerant than muscle of severe permanent diminution of blood-supply, but survives better a temporary deprivation of blood. The evidence is not altogether satisfactory, since, in his experiments, the distribution of the blood to the different tissues and the degree of ischæmia could not be controlled. On *a priori* grounds such tissue characteristics are improbable, and, moreover, the survival experiments reported here do not favour such a belief. In rabbits, if the death of skin were extensive, there was invariably diffuse necrosis of the muscles in the portion covered by surviving skin. In all cases of peripheral gangrene, and in some cases without death of skin, there was also scattered necrosis of muscles. The assumption is reasonable that muscle, as a more highly differentiated tissue than skin, withstands ischæmia less well. Massive death of skin to a higher level than that of subjacent structures can be more satisfactorily explained by the distribution of blood to the tissues, the deeper structures being more generously supplied than the superficial, at least below the knee.

The occurrence of contractures after arterial ligation alone has not previously received attention. Brooks[2, 4] has shown in an admirable series of experiments that the massive fibrosis of muscle which produces rapid development of contractures, such as Volkmann's contracture, is a result not of ischæmia but of a complete obstruction of the venous return from the muscles. His experiments have been confirmed by Middleton,[13] who has demonstrated that the sternomastoid tumour of infants, which gives rise to torticollis, has a similar origin. There is at present a tendency towards the total exclusion of arterial obstruction from the causation of contractures. In the survival experiments on arterial occlusion all animals which did not develop massive gangrene or make a full or nearly full functional recovery, and which survived beyond the second week, developed contractures of the limb. There were ten cases in all. Admittedly peripheral gangrene of the

digits ultimately appeared in all except one, but usually contractures were well established before the appearance of gangrene. The contractures were due to the development of fibrous tissue which slowly replaced the necrotic muscle. The fascia also shared in the fibrotic process. The slow rate of fibrous replacement was no doubt due to the diminished blood-supply to the limb.

It is not suggested that arterial obstruction necessarily plays a part in such conditions as Volkmann's contracture. Obstruction of the main artery of a limb may, however, produce a slowly developing contracture of the limb. Simultaneous ligation of the main vein apparently has no influence on the development of contracture.

CONCLUSIONS.

1. The immediate and late effects of ligating the main artery and main vein of a limb have been studied experimentally.

2. The results of the investigation are not in accord with previous experimental work on the subject. They do not support the current belief that ligation of the main vein diminishes the incidence of gangrene which follows ligation of the main artery.

3. Ligation of the vein at a higher level than arterial ligation *increases* the incidence and extent of tissue death.

4. Theories previously held to account for the supposed benefits of venous ligation have not withstood experimental trial.

I wish to express my thanks to Dr. D. B. Phemister, Professor of Surgery, University of Chicago, for his interest and advice during this investigation.

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ON THE INNERVATION OF THE BLOOD-VESSELS OF THE UPPER EXTREMITY: SOME ANATOMICAL CONSIDERATIONS.

By DONAL SHEEHAN,

DEPARTMENT OF ANATOMY, UNIVERSITY OF MANCHESTER.

The wide adoption of the operation of sympathectomy for vascular lesions has necessitated a more exact knowledge of the anatomy of the sympathetic cord, in particular in the cervical and lumbar regions. The innervation of the blood-vessels to the lower limb has recently been carefully investigated by Woollard,[40] and the operation of lumbar sympathectomy for conditions of arterial spasm in the lower extremities is invariably completely successful, provided that no extensive obliterative changes have superseded the spasm in the arterial wall. Cervico-dorsal sympathectomy for Raynaud's disease, however, although resulting in a definite improvement in the condition of the upper limb, rarely gives a complete recovery. Only too frequently recurrence of the symptoms appears in one or more fingers, usually but not invariably on the ulnar side of the hand. This incomplete result, in marked contrast to that in the lower extremity, has been noted by many surgeons (Davis and Kanavel,[5] Adson and Brown,[1] Telford and Stopford,[36] Royle,[30] Fulton,[9] Hesse[11]), and varied explanations have been advanced to account for this apparent anomaly.

In 1912 Todd[37] described in detail the contribution from the second thoracic nerve to the brachial plexus, and he showed that this communicating branch consisted of both spinal and sympathetic fibres. Later[38] he emphasized its clinical importance in vascular changes produced by pressure on the lowest trunk of the brachial plexus. Kuntz[17] has explained the imperfect results of cervico-dorsal sympathectomy by observing that in a large percentage of cases this intrathoracic ramus arises from the second thoracic nerve and joins the first usually proximal to the origin of the first intercostal nerve. This constitutes a pathway through which sympathetic fibres may leave the sympathetic trunk below the first thoracic ganglion and enter the brachial plexus. Following this suggestion Adson and Brown[2] have operated on a case by the dorsal route with complete extirpation of the stellate ganglion and of the thoracic sympathetic cord as far distal as to include the second thoracic ganglion. They report: "The results obtained in the upper extremities by this procedure are comparable to those obtained with the removal of the lumbar sympathetic ganglia in the lower extremities." It would seem, therefore, that cervico-dorsal ganglionectomy does not often give a complete sympathetic denervation of the hand unless the second thoracic ganglion or its corresponding rami are also included.

Lewis,[26] however, rightly suggests that there can be comparatively few

fibres passing by this channel to the brachial plexus, and he considers that it is most unlikely that spasm of the vessels to the extent of obliteration in all the fingers could ever occur as a response to nerve impulses passing exclusively along this path. From the experimental researches that he has carried out with his co-workers, he comes to the conclusion that sympathectomy will fail to cure the symptoms however wide the excision of nerves, because the real cause of the spasm is peripheral and not central, e.g., a local fault of the vessels. He describes with Landis[27] a case of severe Raynaud's disease of long duration on which right cervico-dorsal sympathectomy was performed. The main trunk of the sympathetic was cut below the second thoracic ganglion and the corresponding rami divided; all nerves joining the inferior cervical ganglion were defined and divided, and the ganglion together with the second thoracic ganglion and the intervening portion of the sympathetic trunk removed in one piece. Following the excision the fingers on the operated side were much warmer than on the opposite side and showed a decidedly lessened tendency to lose their circulation. "It is manifest, however, that the primary cause of spasm remains unchanged in the right hand, and that it does not consist of vasomotor impulses, none of which can now reach the vessels of this hand." Immersion in water at 15° C. produced complete loss of circulation in parts of both hands, though the area involved was less in the right hand than in the left. The relief of the symptoms by sympathectomy is due, according to Lewis, not to removal of abnormal vasomotor impulses, but to removal of normal ones which, even in the normal person on exposure of the body to a cold atmosphere, result in partial vasoconstriction. In Raynaud's disease the closure of the digital vessels is complete. To explain this, Lewis postulates a local fault, which according to him may be of two kinds: (1) Structural changes in the arteries such as intimal thickening; or (2) A more powerful response of these vessels to such a local stimulus as cold.

Lewis's hypothesis does not, however, allow for the extraordinarily perfect results of lumbar sympathectomy for conditions of vascular spasm in the lower limbs. Raynaud's disease is manifestly a diffuse condition; and if a local fault in the digital vessels of the fingers is postulated, why not also for the toes? Furthermore, the intimal thickening or pathological changes in the vessel wall may be regarded, as Lewis admits, as consecutive to the vascular spasm and a diminished blood-flow. Simpson, Brown, and Adson[32] have pointed out that Lewis's evidence is based on cases of advanced degrees of Raynaud's disease in which pathological changes in the vessels had been superadded to the spasm, and therefore is not applicable to the more usual mild cases. They advance evidence in support of the neurogenic origin of the condition. In severe and complicated cases where there are additional vascular changes, even after interruption of all sympathetic fibres, it is admittedly possible for the arteries of certain digits to enter into spasm with resulting cyanosis. It is concluded that in these severe complicated cases there is an abnormal response of the arteries, which is most striking in the fingers that have undergone ulceration of the tip, but this is attributed to *secondary* changes that have taken place in the arteries and arterioles. This point seems of some importance. In the clinical report of the case quoted above

Lewis remarks that "small scales of dry gangrene often occurred on her finger tips."

A third view has been advanced in regard to the frequent imperfect results of sympathectomy. Leriche, who still advocates peri-arterial sympathectomy with good results in simple uncomplicated conditions of vascular spasm,[24] has with Fontaine[25] published some recent deductions from his review of the results of operations on the sympathetic nervous system, whether ganglionectomy, ramisection, or peri-arterial sympathectomy. Such operations are all followed by an active vasodilatation, more or less lasting. This makes itself felt especially distally, but one can find traces of it proximally and often on the opposite side. These results, they hold, can be readily explained if one advances the hypothesis of sensory fibres in the vessels. On this assumption they postulate vascular reflexes, of which they recognize: (1) Peripheral vascular reflexes having their centre in the intramural plexuses; (2) Vascular changes through axone reflexes; (3) Intrasympathetic reflexes which have their centre in the ganglia of the sympathetic trunk; (4) Medullary vascular reflexes; (5) Cerebral vascular reflexes. The vascular changes, write Leriche and Fontaine, are the same after either peri-arterial sympathectomy or operations upon the sympathetic trunk, but in the latter case the modifications in the local circulation are more marked as more pressor fibres have been cut. "There is a quantitative but not a qualitative difference between these two operations."

It is clear that in the operation of cervico-thoracic sympathectomy many post-ganglionic fibres from cells situated in the middle cervical ganglion must remain intact. Kerper,[16] in his experimental work on dogs, found that following excision of the stellate ganglion there remained a number of nerve-fibres in the adventitia, and some in the media. Those in the superficial layers of the adventitia were mainly myelinated, those in the deeper layers of the adventitia and the media were mainly unmyelinated. Kuntz[18,19] believes that at least some of these fibres are sensory in function. Many of the unmyelinated may possibly have been post-ganglionic fibres from the middle cervical ganglion. The assumption of purely sensory fibres to blood-vessels and the possibility of local vaso-reflexes is not new. It has been expressed by many clinical workers in this field, notably Läwen[23] and Friedrich.[8] The evidence has recently been critically reviewed by Stopford.[34] There is no clear anatomical proof of any afferent path from the blood-vessels of the limbs to the ganglionated sympathetic cord. Thus up to the present Leriche's hypothesis has received no definite support from anatomical research, though there is some suggestive evidence in its favour.

Lastly, in explanation of the incomplete results of cervicodorsal sympathectomy, we have the possibility of persistence of the peri-arterial nervous plexus arising from the sympathetic cord close to the origin of the subclavian artery. We do not yet know how far the peri-arterial plexus from the proximal source extends down the upper limbs, or whether there is any marked overlap with the distal source of supply. Woollard[40] has shown in the lower extremity that the overlap is not very great and occurs about the level of the bifurcation of the femoral. In the upper limb it is generally accepted, though unproven, that the proximal source does not reach farther

than half way down the upper arm. Langley[21] and Wiedhopf[39] have found no evidence for the existence of long vasoconstrictor pathways extending along the perivascular tissues as far as the hand, and Dennig[6] also considers that there is none for the vasodilator fibres. It must be admitted that the experimental evidence advanced by these workers against the existence of long perivascular nerve pathways is not in any way conclusive. As regards vasodilator nerves, it seems important to call attention to the work of Kuré and his collaborators from Tokyo[20] who have in recent years suggested a possible new anatomical pathway for vasodilator impulses to blood-vessels through what they term the 'spinal parasympathetic system'. This work is still in progress and awaits further confirmation.

To summarize, we can tabulate the possible factors underlying the incomplete results of cervico-dorsal sympathectomy :—

1. Failure to produce a total sympathetic denervation of the limb, usually from incomplete extirpation of the upper thoracic sympathetic cord.

2. Pathological changes in the peripheral vessels possibly secondary to the vascular spasm, as seems to occur in severe and complicated cases.

3. The possibility of the middle cervical and vertebral ganglia acting as reflex centres : the post-ganglionic fibres from these cell stations remaining intact after cervico-dorsal sympathectomy. This theory necessitates the postulation of afferent vascular nerves.

4. Extension of the peri-arterial nervous plexus as far down as the hand. It will be shown later how in the case of the upper limb the perivascular plexus may not completely degenerate after removal of the stellate ganglion. Whether this has any significance remains to be seen.

All the collected evidence indicates that there is still much to be learnt about the possible anatomical pathways by which nerve impulses can reach the vessels in question, in particular in regard to the upper extremity. It is generally accepted that there are two possible paths for vasomotor impulses to the limbs. " First, a proximal supply, which comes from the lower part of the cervical portion of the sympathetic ganglionated cord and accompanies the subclavian artery in the case of the arm ; and an extension of the aortic sympathetic plexus along the common iliac, external iliac, and femoral arteries in the leg. Secondly, a more distal supply consisting of fine filaments which arise from neighbouring peripheral nerves, and reach the vessels at intervals " (Stopford[34]).

The distribution of the arterial branches of the peripheral nerves in the upper extremity has been carefully studied by Goering,[10] Frey,[7] Kramer and Todd,[15] Hirsch,[12] and more recently by Coates.[4] Anatomical studies directed particularly to the proximal source of vasomotor fibres are singularly lacking, though there are many accounts of the lower cervical sympathetic cord to be found in the works of Potts,[29] Siwe,[33] Jonnesco,[14] Laubmann,[22] Sozon-Jarosevic,[35] and in particular in the excellent and detailed descriptions given by Axford[3] and Hovelacque.[13] Little of anatomical importance can be added to these exhaustive studies, but it is the purpose of this paper to emphasize the significant difference in the anatomical arrangement of the proximal sympathetic supply to the blood-vessels of the upper and lower limbs and to stress certain important surgical relations in regard to the former. A

reinvestigation of the innervation of the blood-vessels of the limbs, based on experimental evidence, is being undertaken. The present investigation has been carried out at the suggestion of Professor J. S. B. Stopford. The lower cervical and upper thoracic sympathetic cord has been carefully dissected on twenty-five sides, attention being particularly directed to the rami communicantes to the roots of the brachial plexus, and to the relationship of nerve-fibres to the subclavian artery.

The most striking feature that became evident in this study has been the great complexity and variability of the branches of distribution from the sympathetic cord and ganglia. A glance at the accompanying sketches (*Figs.* 222–227) will indicate the problem that faces the surgeon in any attempt to produce sympathetic denervation of the upper limb. Some of the rami communicantes arise from the anterior and posterior aspect of the ganglia

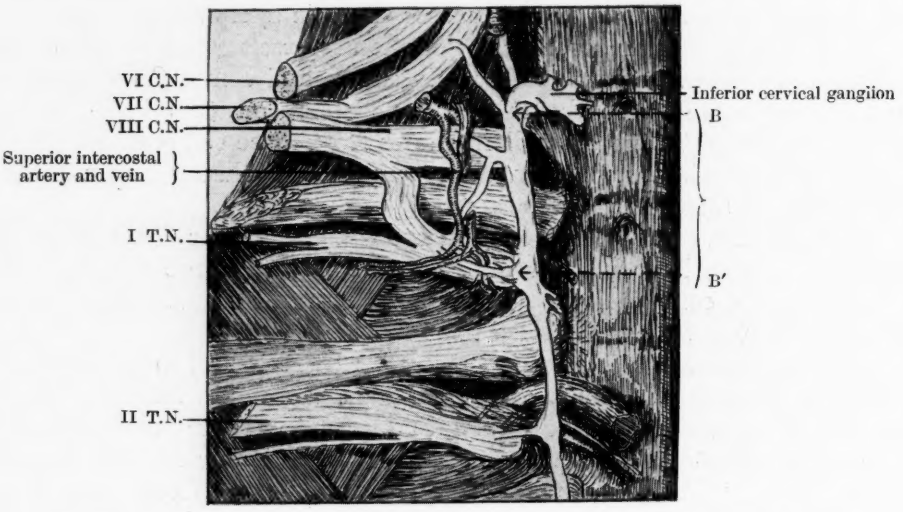


FIG. 222.—Right side of body. Showing the distance between the first thoracic ramus and the inferior cervical ganglion. Distance B–B′ = 1 in. Note the relation of the superior intercostal artery and vein to the sympathetic cord; the vein lies medial to the artery.

and run for a course of less than $\frac{1}{2}$ cm., a course often entirely hidden from view. Again, the number and exact site of origin of these branches is variable and their relationship to the superior intercostal artery very intimate. The distinction drawn by Axford between white and grey rami is not at all so clear at operation as one would be led to expect from anatomical dissections. It seems evident that in order to ensure complete sympathetic denervation of the upper limb, the operation of ganglionectomy (inferior cervical and the upper two thoracic) is a much surer and more practical proposition than any attempt at cervico-thoracic ramisection.

A second point of some surgical importance was observed soon after the commencement of this study. The contribution of the second thoracic nerve to the brachial plexus, and the existence through this communication of a pathway by which sympathetic fibres can reach the upper limb from well below the inferior cervical ganglion, has been emphasized by Kuntz. But

this is not the only possible loophole in an incomplete cervico-dorsalectomy. The grey rami to the first thoracic nerve frequently arise some distance below the inferior cervical ganglion, even in those cases where the latter is partially or completely fused with the first thoracic ganglion. *Fig.* 222 demonstrates this point very clearly, the distance separating the lower end of the inferior cervical ganglion and the rami communicantes to the first thoracic nerve being as much as one inch. In such cases this is an additional and very important source of sympathetic fibres to the upper extremity. It is essen-

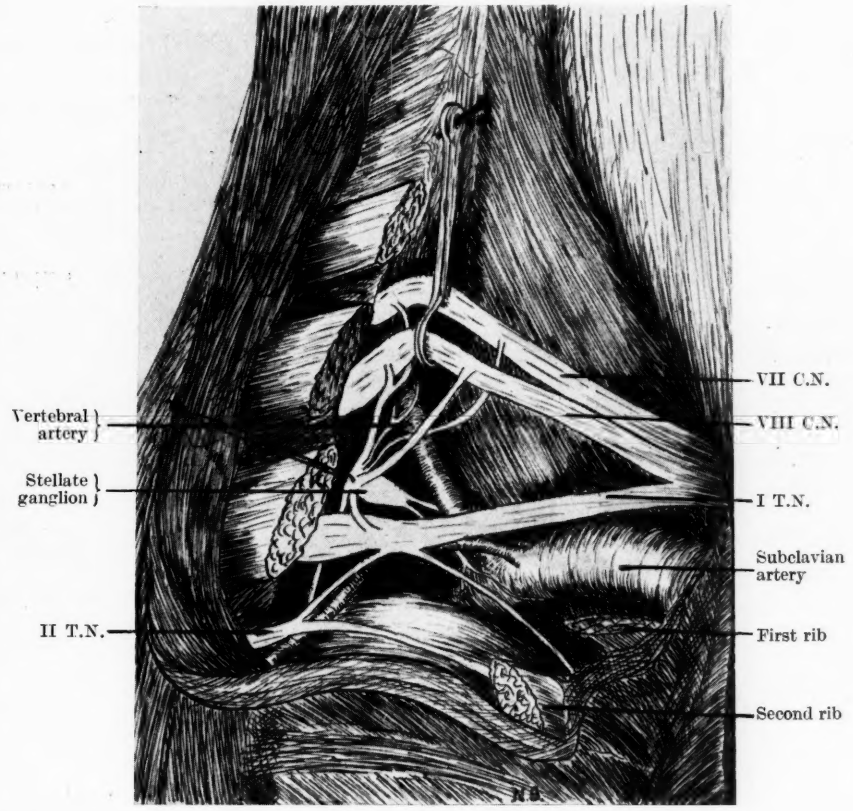


FIG. 223.—Right side. Posterior approach. Portions of first and second ribs removed. Note the position of the stellate ganglion between the eighth cervical nerve and the first thoracic nerve.

tial therefore at operation to remove the upper thoracic cord as far caudally as to include the second thoracic ganglion. By this means only can all the pre-ganglionic fibres to the upper limb be securely cut. In order to ensure this low excision, the dorsal route for the operation is essential, removing a portion of the first rib and preferably a part of the second (*Fig.* 223). This point is mentioned, as the anterior route is still practised by some surgeons in this country.

In performing this posterior approach the anatomical relationship of the sympathetic cord to two structures in particular appears important—namely,

the superior intercostal artery and the thoracic duct. The superior intercostal artery has been preserved in several of the dissections and is illustrated in *Figs.* 222, 224–226. It arises above the inferior cervical ganglion from the posterior aspect of the subclavian artery. It arches laterally and dorsally over the upper part of the ganglion, gives off its deep cervical branch, and then itself continues downwards along the lateral border of the inferior cervical and first thoracic ganglia, crossing over ventrally the eighth cervical and first thoracic nerves in this order. It frequently gives off a branch from its medial aspect, which passes posterior to the intervening sympathetic cord between the first and second thoracic ganglia. Not only is it important to recognize and secure this artery, or one of its branches, at the time of operation,

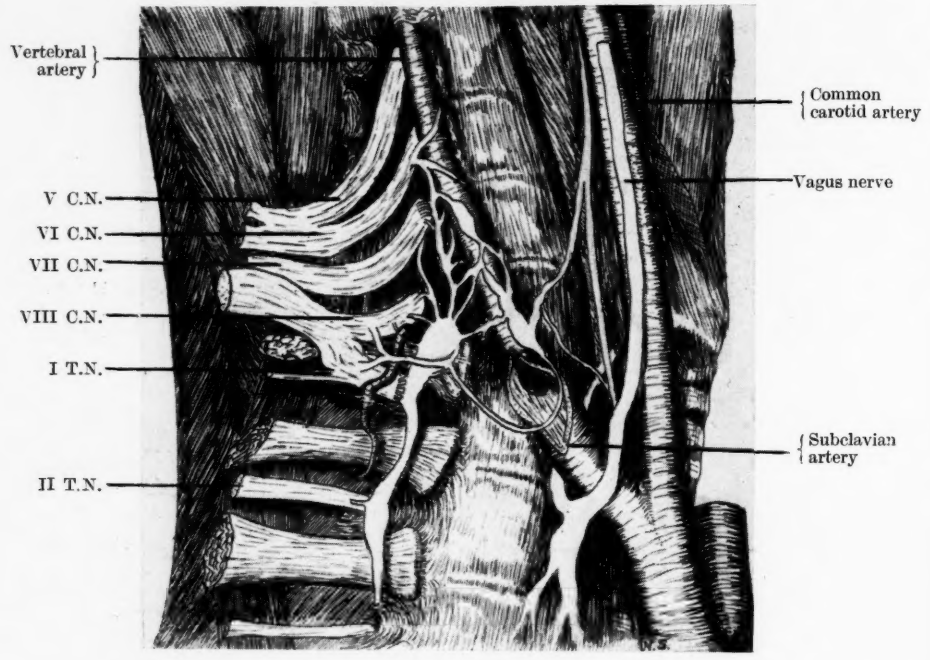
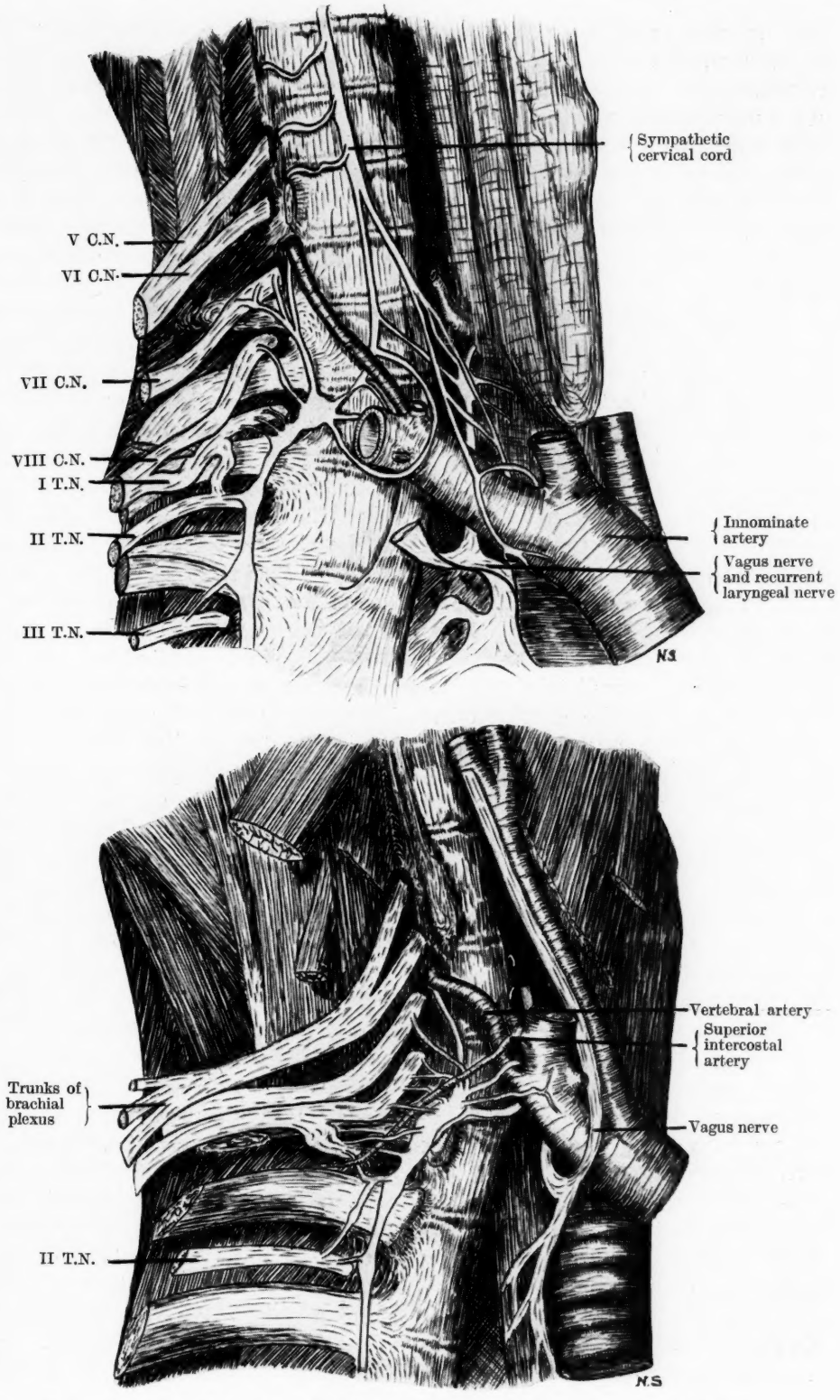


FIG. 224.—Right side. Anterior tubercles of transverse processes of sixth and seventh cervical vertebræ removed to show the vertebral plexus. The relationship of the superior intercostal artery can also be seen.

but it also may serve as a very helpful guide to the exact position and direction of the upper thoracic cord. The accompanying vein may be represented as two venæ comites, but more frequently it passes down medial to the artery, lying between the latter and the sympathetic cord (*see Fig.* 222). In this position it is therefore in greater danger of being damaged than the artery.

The important relationship of the inferior cervical ganglion to the thoracic duct has been emphasized by Minkin[28] in relation to cervical sympathectomy by the anterior approach. By this route the bend of the thoracic duct must be defined and carefully avoided as it hooks round on the ventral aspect of the stellate ganglion, from which it is separated by a thin layer of fatty tissue traversed by numerous small veins. The arch of the thoracic



FIGS. 225, 226.—Right side. Showing two typical appearances of the inferior cervical ganglion and its branches.

duct, according to Minkin, may lie higher or lower than, or completely lateral to, the stellate (or inferior cervical) ganglion. The lymphatic duct and its tributaries may closely simulate small veins, and may occasionally be ligatured and divided accidentally, under this impression. By the posterior approach for cervico-thoracic sympathectomy it would appear that the thoracic duct is very rarely in danger of being involved. The upper thoracic sympathetic cord is always reached first, the lymphatic duct, from this aspect, lying deeper. Furthermore, at this level the sympathetic cord is inclining backwards to lie directly on the ventral surface of the ribs, whilst the thoracic duct is arching forwards and therefore away from the stellate ganglion.

The contribution from the middle cervical ganglion to the brachial plexus consists of rami communicantes to the fourth and fifth cervical nerves, and in some cases it was observed to give a twig to the sixth. Occasionally these branches arose direct from the sympathetic cord, either below or immediately above the middle cervical ganglion. Much of the difference in opinion concerning the incidence of this ganglion and its exact position appears to be due to a confusion in nomenclature rather than to any particular difference in observation. The usual description is that the middle cervical ganglion, when present, lies opposite the level of the sixth cervical vertebra immediately anterior or posterior to the inferior thyroid artery, and that it is formed by the coalescence of the fifth and sixth ganglia. This is frequently referred to as the 'thyroid' ganglion. But there is a second—and from the present study it would appear a more constant—ganglion, situated on the lower cervical chain immediately ventral to the root of the vertebral artery. It is connected with the inferior cervical ganglion usually by two short stems which encircle the vertebral artery. This has been called the 'vertebral' ganglion by some, and by others the 'intermediate' ganglion (Jonnesco). From the present study one or other of these ganglia was almost invariably present, though occasionally very small in size; and it seems better to follow the description as given by Axford and consider them both as constituting the middle cervical ganglion, and to distinguish them by the terms 'high' and 'low' types respectively. In those cases where they occur together, the middle cervical ganglion is obviously double. Laubmann divides the arrangement of the cervical sympathetic cord into five main types, and by far the commonest was that in which the superior, middle (either 'high' or 'low' type), and inferior cervical ganglia were all present and distinct. The high type of middle cervical ganglion is crossed, usually anteriorly, by the inferior thyroid artery just where the latter loops downwards to meet the recurrent laryngeal nerve. In addition to the rami communicantes described above, the ganglion gives off the middle cardiac nerve, branches along the inferior thyroid artery, and occasionally, as seen in *Fig.* 227, a communicating branch to the phrenic nerve. The ansa subclavia may come from the 'high' or 'low' types of the middle cervical ganglion or from the sympathetic cord itself.

The inferior cervical ganglion is partially or entirely fused with the first thoracic ganglion in about 80 per cent of cases (Testut), the united mass being known as the 'stellate' ganglion or ganglion of Neubauer. All degrees of this fusion can be seen (*Figs.* 224–226). A complete union would appear from the present study to occur in only about 50 per cent. An important

alteration in the direction of the sympathetic cord occurs at the inlet to the thorax. In the lower part of the neck the cord lies in a direction downwards and with a slight inclination medially. This ends at the so-called 'vertebral' or low type of middle cervical ganglion, and from here the inferior cervical ganglion turns backwards over the transverse process of the seventh cervical vertebra and over the neck of the first rib. The continuation of the upper thoracic cord is at first backwards as well as downwards and laterally. It then runs along a line drawn at right angles to the long axis of the ribs about half an inch from the costovertebral articulations.

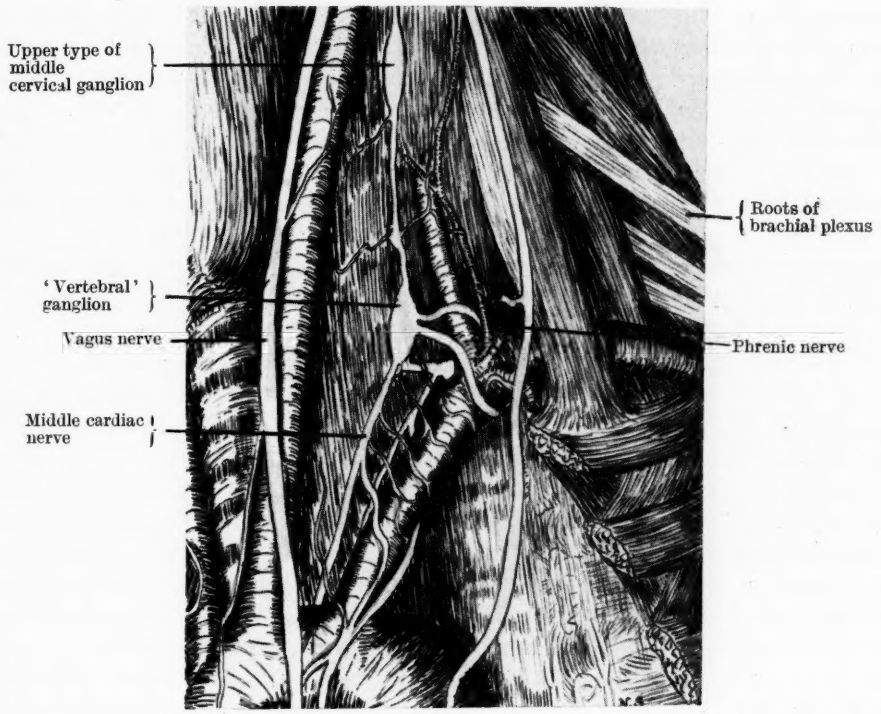


FIG. 227.—Left side. Showing the appearance before removing a portion of the subclavian artery. Note the presence of both 'high' and 'low' types of middle cervical ganglion. There is also a connection between the sympathetic and the phrenic nerve. This figure illustrates the extensive peri-arterial plexus around the origin of the subclavian artery.

The stellate ganglion lies in a niche posterior to the pleura and to the subclavian artery and sometimes to the origin of the vertebral artery, and anterior to the transverse process of the seventh cervical vertebra and the neck of the first rib. In the high position it may actually present a groove or depression on its anterior aspect, in which the vertebral artery rests. The stellate or inferior cervical ganglion is constantly to be found between the eighth cervical and first thoracic nerves, which it crosses on their ventral surface. In size the inferior cervical ganglion has been found never to measure less than 1 cm., and in those cases where it is united with the first thoracic ganglion its length may exceed 2 cm. A simple idea of its thickness can be obtained by remembering that it is usually at its widest part not very much

less than the eighth or first thoracic nerves which it is crossing. No useful purpose is fulfilled by attempting to assign any particular form or shape to the ganglion. It will be seen from the accompanying figures that occasionally it is truly stellate, but more often triangular or even an elongated inverted cone (*Figs.* 224–226).

The branches of distribution from the present study of the stellate ganglion can best be briefly summarized as follows :—

1. **Rami Communicantes.**—

a. Superficial or Direct Branches.*—To fifth, sixth, seventh, and eighth cervical and first thoracic nerves. These all took origin from the upper and lateral aspects of the ganglion. Often they were double or divided into two or three branches as they reached the spinal nerve. They usually passed behind the superior intercostal artery except those to the eighth cervical nerve, which frequently crossed on the ventral aspect of the artery. For a detailed account of the exact course and relationship of each of these rami, the excellent paper by Axford should be consulted. The particular significance of the grey rami to the first and second thoracic nerves has already been emphasized.

b. Deep or Indirect Branches from the Vertebral Nerve.—To fourth, fifth, and sixth cervical nerves.

2. **Nervus Vertebralis** arose from the superior aspect of the ganglion. It was very constant. It followed the vertebral artery on its lateral side, and with the latter entered the vertebral foramina in the cervical transverse processes. As it passed upwards it gave off important twigs to the vertebral plexus around the artery, but its largest branches were given off to the sixth, fifth, and often fourth cervical nerves as they crossed behind the vertebral artery. *Fig.* 224, in which the anterior tubercles of the transverse processes are shown removed, demonstrates this very clearly. These 'deep' rami communicantes would of course degenerate in a cervico-thoracic ganglionectomy, but the close association of the nerve with the vertebral artery would make it extremely difficult to ensure its division in a ramisection.

3. **Ansa Subclavia** (*Ansa Vieussens*)† arose above from the middle cervical ganglion or the lower cervical sympathetic cord, crossed over the first part of the subclavian artery, and wound round the lower aspect of this in a long course to reach the anterior surface of the inferior cervical ganglion. Because of its attachment to the ventral aspect of the ganglion, and on account of its very close relationship to the subclavian artery, it is in great likelihood of being missed in any operation of ramisection by the dorsal route. It was frequently double, and may form a connection with the phrenic and with the recurrent laryngeal nerves (*see Fig.* 227). This has been emphasized by Shawe,[31] who found it present in 68 per cent of the specimens investigated. It distributes branches to the peri-arterial nerve plexus around the subclavian artery.

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* Following the method of description as given by Hovelacque.

† Hovelacque has pointed out that Vieussens (1684) actually described a communicating branch uniting the inferior cervical and the first thoracic ganglia, in front of the subclavian artery.

4. **Direct Vascular Twigs** to the subclavian artery and its branches, particularly the vertebral.

5. **Short Stout Communicating Branches** surrounding the origin of the vertebral artery, and uniting on its ventral aspect in the 'vertebral' or 'low' type of middle cervical ganglion or in the cervical cord itself.

6. **Inferior Cardiac Nerve** arose from the supero-medial aspect of the inferior cervical ganglion, or frequently in common with the ansa subclavia. In the present study it passed most commonly, both on the right and the left side, along the lower and posterior aspect of the subclavian artery, to the deep cardiac plexus. It nearly always formed a plexus of communication, and in some cases actually blended with the middle cardiac nerve and the recurrent laryngeal nerve. This plexus surrounds the origin of the subclavian artery, particularly posteriorly, to which it gives numerous branches (*see Fig.* 227). This is only illustrated by Hovelacque (*Plate XCIX*, p. 676) and by Jonnesco (*Fig.* 1, p. 92). In this connection Jonnesco gives the following very significant passage: "Dans leur long trajet, les nerfs cardiaques s'anastomosent entre eux, avec les rameaux cardiaques du pneumogastrique, et avec les rameaux du laryngé supérieur et inférieur. Quelquefois, ils s'approchent l'un de l'autre en changeant de fibres, directement, et forment un plexus. D'autres fois, au niveau de leur contact on trouve un ganglion." The cardiac nerves often present along their course one or more ganglia. According to Jonnesco three are more or less constant, one at the point where the superior cardiac nerve anastomoses with the middle, a second along the course of the superior cardiac nerve, and a third on the course of the middle cardiac nerve in its thoracic portion. It is an important and as yet unanswered question how far this intimate plexus between the sympathetic cardiac nerves and the vagus (and occasionally the phrenic) enters into the innervation of the blood-vessels of the upper extremity. Nor can the presence of these small ganglia in this plexus which surrounds the first part of the subclavian artery be entirely overlooked. There appears here to be a possible difference in the anatomical arrangement of the vascular nerves in the upper and lower extremities.

In conclusion it would appear that if one could be certain that the perivascular plexus around the subclavian artery is entirely sympathetic, and if one can exclude the possibility of vascular reflexes through remaining sympathetic ganglia, then it should be possible to denervate completely the sympathetic supply of the upper limb by removing the stellate ganglion and the upper thoracic cord as far down as to include the second thoracic ganglion. By this means all the pre-ganglionic fibres should be severed.

SUMMARY.

1. The difference in the upper and lower extremities in the operative results of sympathectomy for vascular lesions has been emphasized.

2. The various theories that have been advanced to explain this difference have been reviewed.

3. The anatomical arrangement of the proximal source of vascular nerves to the upper extremity has been investigated on twenty-five sides.

4. Certain important surgical and anatomical features in relation to the operation of cervico-dorsal sympathectomy are recorded.

5. The dorsal route is the only possible method of ensuring complete sympathetic denervation of the upper limb, and the operation of ganglionectomy would appear to be a much surer and more practical method than that of ramisection.

My thanks are due to Professor J. S. B. Stopford for allowing me to carry out this work in his department, and above all for his kind help and advice. For permission to examine their clinical cases, both before and after operation, I am indebted to Professor Telford and Professor Stopford. I also wish to express my most sincere gratitude to Mr. N. Shtetinin, third-year medical student, for the excellent illustrations which he has so kindly made from my dissections.

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A REPORT ON THE STRANGEWAYS COLLECTION OF RHEUMATOID JOINTS IN THE MUSEUM OF THE ROYAL COLLEGE OF SURGEONS.

By R. LAWFORD KNAGGS,

With Photomicrographs by G. H. RODMAN.

PART III.

THE PATHOGENESIS OF GOUT.

I. THE ARTHRITIC PHENOMENA ASSOCIATED WITH GOUT.

In Part I attention was drawn to the similarity of many of the signs present in gouty joints to those which distinguish osteo- and rheumatoid arthritis, and to the impossibility of distinguishing specimens of the latter disorders from those of gout, without the help of a urate of soda deposit. At the same time the question of the toxic origin of these arthritic changes was also considered.

Now, in joints that have been affected by gout for a number of years there are usually found 'mixed' arthritic changes, such as lipping, grooving, eburnation, and bony and fibrous ankylosis, with microscopic evidence of fibrous degeneration of bone and cartilage. This association of hypertrophic and degenerative signs is present in almost every one of the Strangeways specimens of gouty joints, and is probably to be explained by the steady diminution of the vitality of the joint tissues as the disease progresses.

Gout is usually met with in fairly healthy people, and its onset, as a rule, occurs in the middle period of life, before the bodily powers have shown a tendency to wane. Therefore, apart from the inflammation set up by the crystalline deposit, the arthritic signs may be expected to take on the osteo-arthritic form. Indeed, nearly every specimen of gout in the Collection presents hypertrophic bony phenomena. These overgrowths, though they may undergo internal changes, are practically permanent. As health declines, and the joint becomes further damaged owing to the increase of the gouty deposit and the associated inflammation, degenerative changes set in. These are of the same nature as those met with in rheumatoid arthritis, and are characterized by fibrocellular degeneration of articular bone and cartilage, in which the hypertrophic formations are not only involved, but are, often, the first to suffer.

The inflammation set up by the gouty deposit, however, introduces an element of confusion. It is probably present during the evolution of the hypertrophic formations, and may account for the occasional development of bony ankylosis in the large joints, as well as in the small ones of the hands

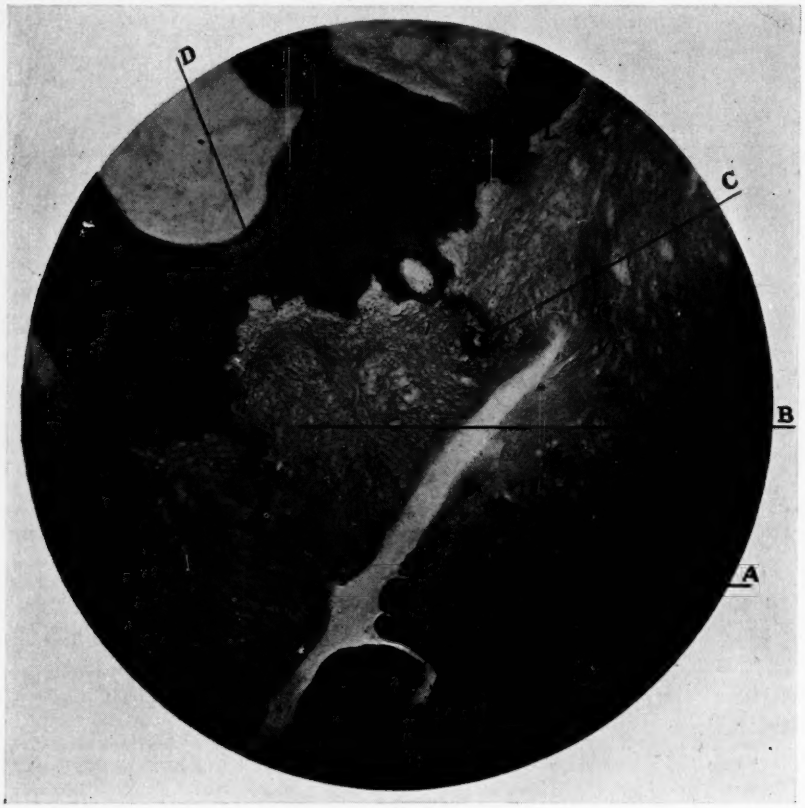

FIG. 228.—Charcot's disease. From an elbow; section through the capitellum showing fibrous degeneration of bone *en masse*. A, Degenerating and disintegrating articular cartilage; B, Massive degeneration of sclerosed bone into a very delicate network of fibrous tissue, permeated by very small spaces—probably cavities from which bone cells have disappeared, while others—larger and in groups—suggest early fat formation; C, Remains of osseous débris; D, Irregular bone edge showing continuity in many places with the fibrous tissue. (*Specimen* S.C. 90.) (× 75.)

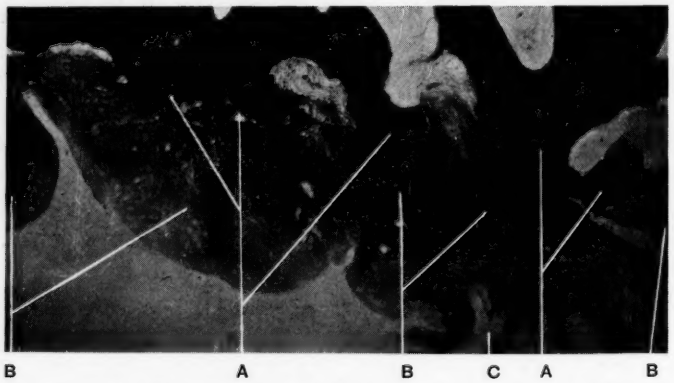

FIG. 229.—Charcot's disease. Section from articular surface of the humerus—from the same elbow as *Figs.* 228 and 234. A, Degenerating superficial portions of sclerosed bone; B, Fibrous covering, evidently largely produced by fibrous degeneration of bone; C, Fibrous villi. (*Specimen* S.C. 90.) (× 50.)

and feet, without invoking the peculiar activity of the cartilage cells, which precedes bony ankylosis in rheumatoid arthritis. It may also be expected to intensify the inflammatory changes usually associated with rheumatoid arthritis, and by its damaging influence on the joint tissues to predispose to the onset of degeneration.

It is not surprising that inflammation in gout is more acute and widespread than in rheumatoid arthritis, for urate of soda is deposited far beyond the articular surfaces. It may be found in the capsule, the bursæ, the tendons, the periosteum, and even in the trabeculæ; and deposits may occur in the medulla well outside the limits usually involved in rheumatoid arthritis. In the latter situation they may be of considerable size. Degeneration, and absorption of bone by granulation tissue, may even lead to the formation of cavities in the cancellous tissue, and these may be packed with urate crystals (S.C. 74).

In nearly every instance the gouty deposit is probably the first pathological lesion to develop. Yet this is not always the case, and the Collection contains one example from an old woman aged 85 who had been a typical case of rheumatoid arthritis for many years—certainly twenty, and probably more; both knee-joints show pronounced evidence of that condition, but in the left there are a few patches of urate of soda in places (S.C. 83).

II. THE MICROSCOPICAL CHARACTERS OF THE GOUTY DEPOSIT.

Urate of soda crystals seem to give rise to little or no irritation when in small collections in the cartilage; but in the capsule, synovial membrane, and the bone medulla, they do. Inflammatory cells are often present in the neighbourhood of deposits in the synovial membrane and capsule, and uratic masses are frequently found in the medulla in the centre of an area of granulation tissue. Within such an inflammatory focus trabeculæ, or portions of trabeculæ, are sometimes undergoing fibrous resolution.

In synovial villi small deposits may have a circle of elongated connective-tissue cells around them, suggesting an attempt at encapsulation. (*Fig.* 230.)

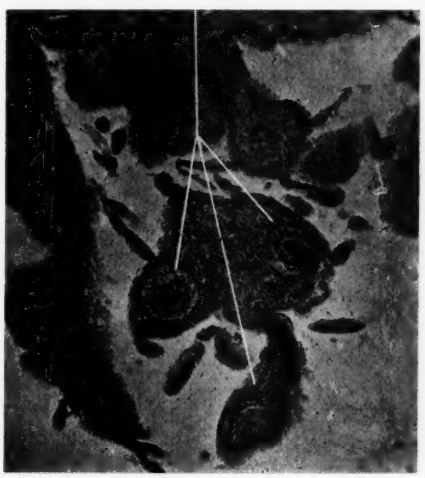

FIG. 230.—Gouty deposits of urate of soda in synovial villi. (*Specimen* S.C. 65.) (× 50.)

Giant cells are not infrequently present in the immediate neighbourhood of deposits, and often several may be closely applied to one. An interesting appearance was seen in connection with the remains of the articular cartilages of an astragalo-calcanean joint from a woman aged 76, in whom both bones were fused. The articular cartilages lay in the midst of fat medulla occupying the spaces of a very atrophic cancellous tissue; they were undergoing fibrocellular degeneration with the production

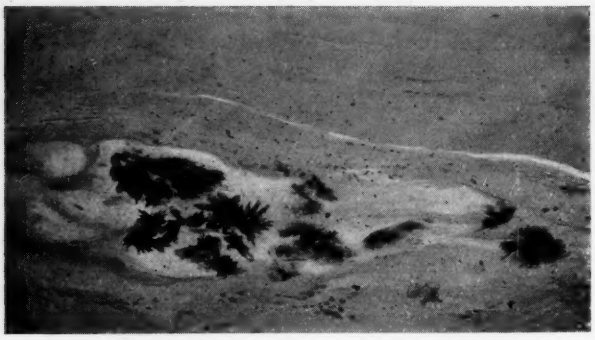

FIG. 231.—Crystals of urate of soda in the capsule of an elbow-joint. They have the usual yellow-brown colour characteristic of urates, more particularly at the thicker parts of the rosettes. (*Specimen* S.C. 67.) (× 85.)

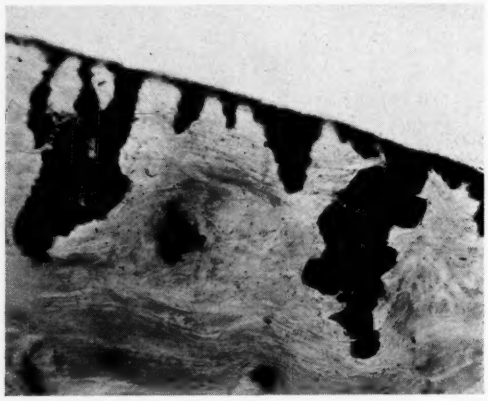

FIG. 232.—Thick deposit of urate of soda crystals in, and on the inner surface of, the capsule of an elbow joint. Owing to the thickness of the deposit, it presents a dark brown colour. Under a high-power its component crystals are easily seen. (*Specimen* S.C. 67.) (× 50.)

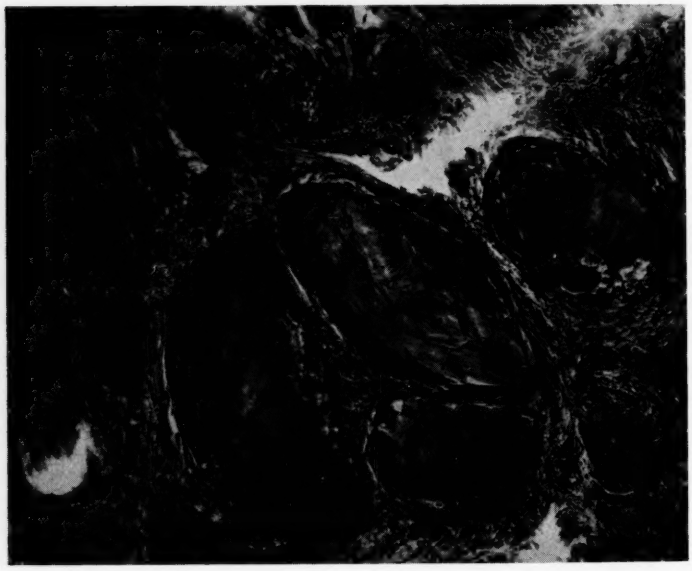

FIG. 233.—Deposits of urate of soda in an inflammatory mass which has penetrated a patella from its dorsal surface and appears to have isolated a portion of the bone. The deposits tend to become encapsulated and have a number of giant cells on their edges. The crystalline texture of one of the large masses is seen in the photograph. From a woman, aged 76. (*Specimen* S.C. 25—2.) (× 100.)

of large numbers of giant cells; some urate of soda deposits of good size, which had possibly been liberated by the dissolution of the cartilage, were surrounded by giant cells, which in one instance were so numerous that the mass was encircled with a row of cells like a necklace of beads (S.C. 81—1). Under the microscope, urate of soda deposits may appear as well-defined, structureless, white or grey patches. In the superficial layers of the cartilage these may be very small, and sometimes small chains of granules may be seen near them in the same layers—possibly these granules are not crystalline. Larger deposits form white masses having a somewhat hazy and granular look. On careful focusing with a high power a delicate fibrillar network may be detected in them in some instances. The needle-like crystals of which the deposits are composed can sometimes be distinguished, but as a rule they cannot, probably because the decalcifying process has caused some change in their composition, or has dissolved them out to some extent. In some instances the crystals may show the yellow-brown colour typical of urates. This is more likely when the deposit is copious and thick. Sometimes beautiful star-shaped masses of crystals may be seen, in which the colour is most pronounced at the nodal or thickest parts, whilst the rays show little of it. In others, a long tract of dark-stained material may infiltrate the surface layers of some joint structure such as capsule or articular cartilage, and its crystalline nature may be revealed by a high power.

THE PATHOGENESIS OF CHARCOT'S DISEASE.

I. THE HISTOLOGY OF CHARCOT'S JOINTS.

When the literature of Charcot's joint disease was investigated a few years ago very little information was found about the histology. Mott—apropos of fractures in tabes—had shown that the Haversian canals were irregularly dilated and filled with fat (*System of Syphilis*, iv, 366–370), and Targett had pointed out that the ragged-looking membrane, presenting numerous villous processes and polypoidal masses, which lined the interior of the joint cavity, and was more or less continuous over the eroded bone-ends, was of fibrocartilaginous structure, and that the polypoid bodies were formed by cartilage, in part hyaline, and in part fibrous, whilst calcification was going on in the centre (*Pathol. Soc. Trans.*, xlii, 270). Neither of these observations dealt with the microscopical changes in the articular ends of the bones, and they leave untouched the vague impression that the erosion of the bone-ends is due to mechanical attrition of parts from which trophic influence has been cut off.

Nine cases of Charcot's disease are represented in the Strangeways Collection, and there are numerous microscopical sections obtained from as many as thirteen joints. The study of these revealed a number of interesting facts.

A description of the histology of Charcot's joints would, for the most part, be simply a repetition of the account of the degenerative changes in rheumatoid arthritis. The articular cartilage, in a very early stage of its degeneration, shows a villous surface, numerous foci of aggregated cells, and

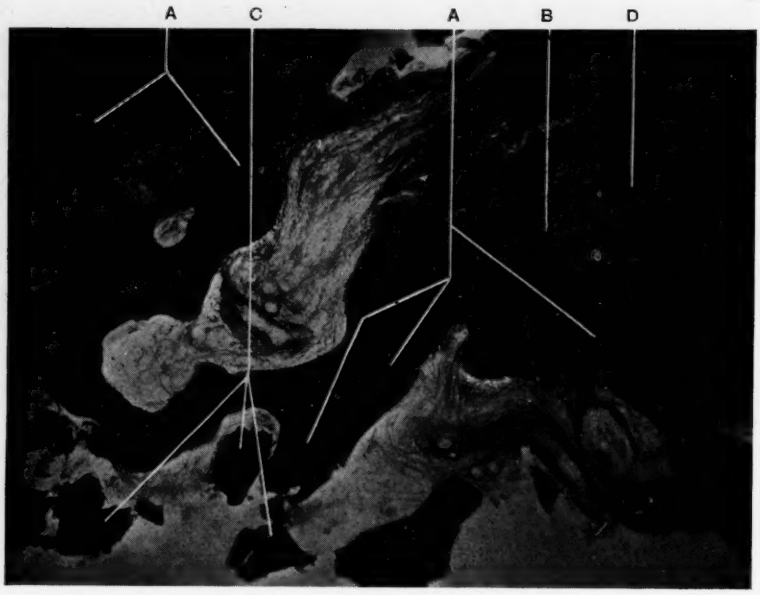

FIG. 234.—Charcot's disease. Section of the articular surface of an ulna—from an elbow. No history. **A**, Degenerating trabeculæ; **B**, Fibrous covering formed from fibrous degeneration of bone; **C**, ? Artefacts, which would be easily caused; **D**, Portion of a trabecula in the fibrous covering in process of resolution. (*Specimen* S.C. 90.) (× 50.)

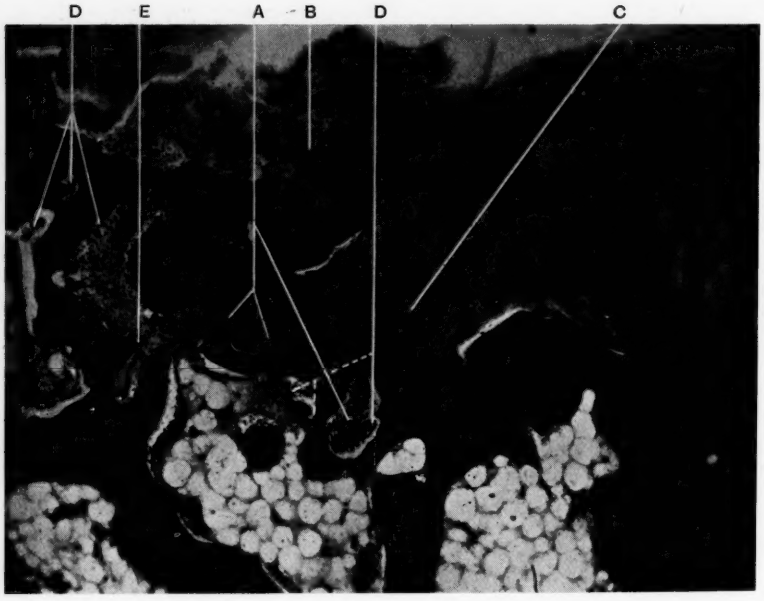

FIG. 235.—Charcot's disease—an early condition in a knee. The photograph shows the only part in a section through an external femoral condyle where characteristic changes occurred. The section included a small quiescent marginal osteophyte containing fat marrow. **A**, Degenerative spaces in bone; **B**, Atrophic cartilage; **C**, Degenerating and atrophying cartilage merging in indistinct bone; **D**, Giant cells; **E**, Compound space communicating with the marrow. (*Specimen* S.C. 93—2.) (× 50.)

spaces in process of formation. It is easy to understand its speedy disappearance, and often the only trace of it may be a mere strip of fibrous tissue.

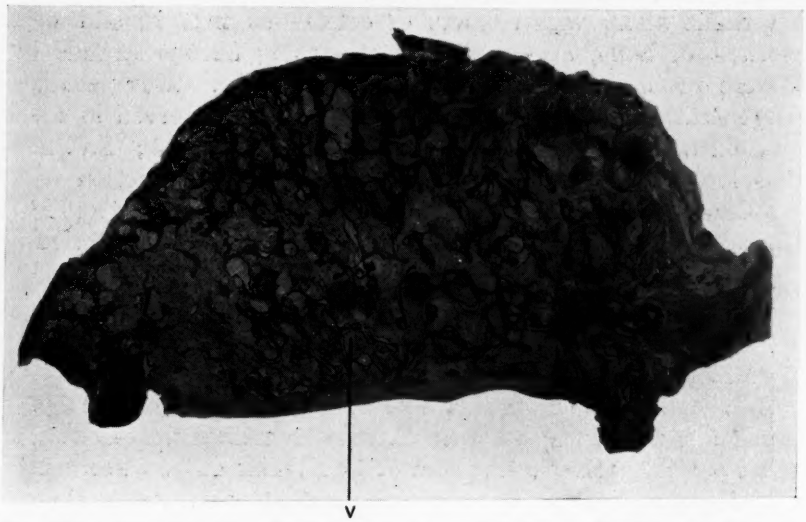

FIG. 236.—Charcot's hip. Section from an eroded femur showing a curious appearance presented by the atrophying bone. V indicates the part which is enlarged in *Fig.* 237. (*Specimen* S.C. 86.) (× 6.)

In the articular ends of the bones the essential change is fibrous degeneration. There is the same formation of spaces in the bone and in the cartilage, the same resolution of those structures into fibrocellular tissue, and the same development of giant cells, as in rheumatoid arthritis. But there is this difference in Charcot's disease—the degenerative changes are more pronounced and more widely spread. Not only are large blocks (microscopical) of fibrous tissue seen in places that have previously been occupied by bone or cartilage, but the evidence of degeneration and resolution extends to much greater depths. Indeed, degeneration sometimes seems to take place *en masse*.

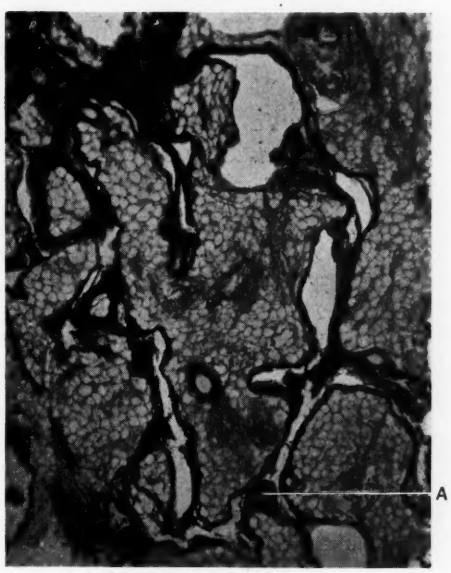

FIG. 237.—Charcot's hip. The illustration shows a portion of *Fig.* 236 (V). The degenerating trabeculæ form a network of anastomosing channels owing to the disappearance of their central parts. The walls of the channels show traces of lime (hæmatoxylin stain), and at A a persisting portion of bone projects into the lumen where three channels join. (× 40.)

There can be no doubt that this degeneration of cartilage and bone is responsible for the rapid erosion of the articular ends of the bones, which is one of the most striking features of Charcot's disease.

An interesting variation in the form of fibrous degeneration of bone was first noticed in a section from a Charcot's

hip; a network of trabeculæ had been converted into a series of anastomosing channels owing to the degeneration, and disappearance, except for some fine fibrils, of their central portions. The walls of the channels were formed of fibrous tissue along which traces of calcareous matter, and occasionally small portions of bone, appeared. Anastomosing fibrous strands in the fat medulla were probably relics of vanished trabeculæ. This peculiar type of fibrous degeneration of the bone trabeculæ was also observed in several cases of rheumatoid arthritis (S.C. 23—1; S.C. 27—1; S.C. 32; S.C. 35—14).

It has long been recognized that the extensive destruction which takes place in a Charcot's joint is induced by the withdrawal of a trophic function. This is an acknowledgement of the part that vitality of the articular tissues

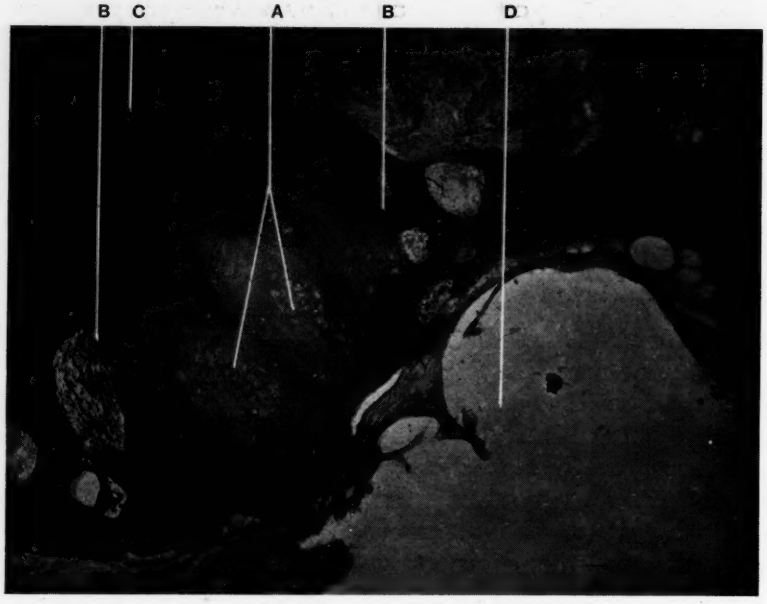

FIG. 238.—Charcot's disease. Section from the articular surface of the head of the humerus—from the right shoulder of Philip H., age 50. A, Degenerating areas in a sclerosed trabecula; B, Spaces which are in process of extension by continued degeneration of portions of their margins; C, Sclerosed bone, which (outside the picture) is covered by a considerable thickness of fibrous tissue resulting from bone resolution; D, Cystic space, whose bony margins are recruiting the fibrous tissue which lines it. (*Specimen* S.C. 89—2.) (× 50.)

plays in this affection. The similarity of the microscopical changes suggests that the only etiological difference between rheumatoid arthritis and Charcot's disease is, that in the latter the innervation of the joint has been impaired. The much greater destruction in the case of a Charcot's joint may be explained by the more extensive degeneration, and by the absence of pain permitting free movements.

It might be argued that Charcot's joint disease is simply rheumatoid arthritis in a patient who suffers from tabes; but the nerve defect, associated with the more widely spread degeneration, and consequent inordinate destruction, produces a combination of symptoms so unique that it would be absurd to think of grouping the two conditions together. It is sufficient if the similarity of the microscopical changes is recognized and the relationship admitted.

II. OSTEOPHYTES AND CAPSULAR OSTEOPHYTES IN CHARCOT'S DISEASE.

It was the remarkable development of osteophytes that caused the early controversy as to the nature of Charcot's joints, and furnished the best argument for those who regarded the affection as osteo-arthritis in a tabetic. But the formation of osteophytes seems incompatible with the rapid degeneration of the articular bone and cartilage that takes place in these cases. There are, however, two kinds of osteophytes—those growing in connection with

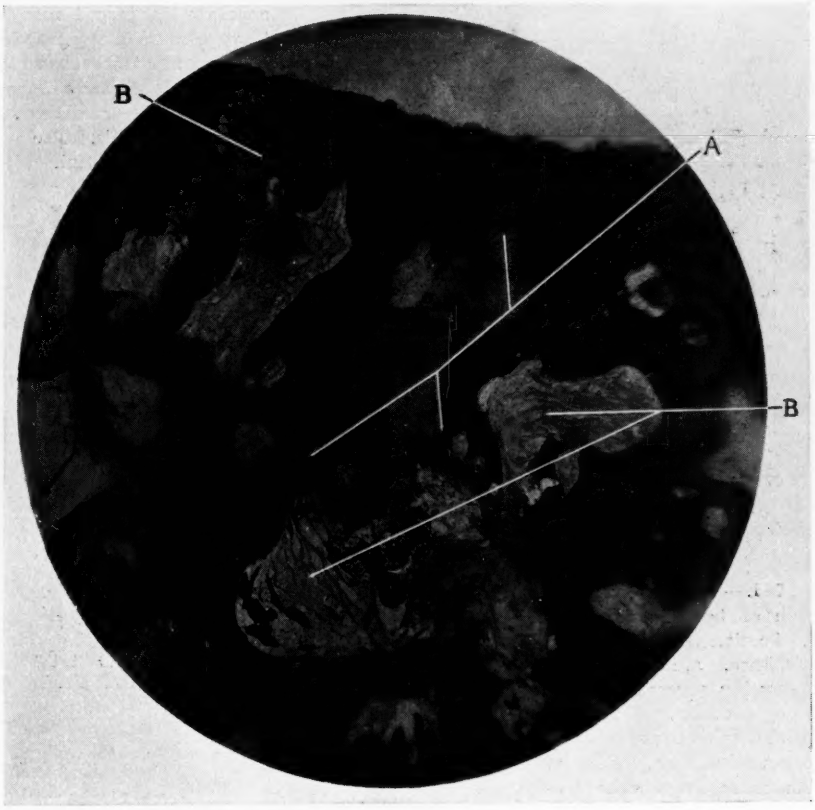

FIG. 239.—Charcot's disease. Section from a knee—not in the Collection, but described particularly in the Strangeways MS., *Fig.* 104A. It shows widespread degeneration of the articular surface. A, Massive degeneration of bone in a sclerosed and eburnated area, fibrous tissue becoming evident; B, Advanced stage. Spaces containing coarse fibrous tissue, resolving fragments of bone, and small quantities of small fat globules. (*Specimen* S.C. 85.) (× 40.)

the articular cartilage, and those forming in the capsule. The latter are numerous in Charcot's disease, and as they have a different mode of origin from articular or marginal osteophytes, the incompatibility just mentioned may be capable of explanation.

The result of a special examination of the Charcot's joints in the Strangeways Collection, and in the Hunterian Museum, showed that though there was no clear evidence of the presence of osteophytes of the articular surface or margin, yet the extravagance of the new bony formations made it impossible to be sure that they did not exist.

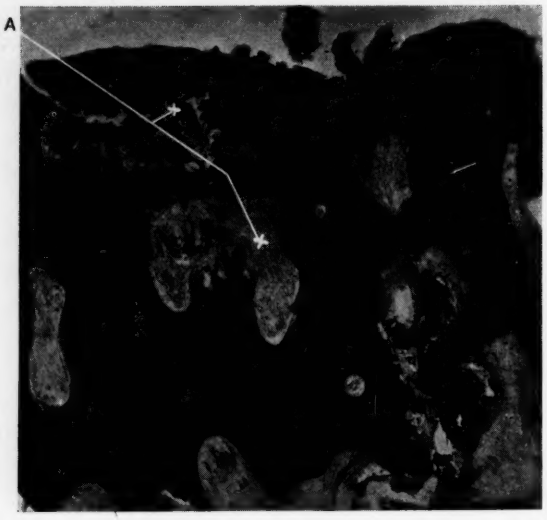

FIG. 240.—Charcot's disease. From the same joint as *Fig.* 239. Degenerative changes in sclerosed and eburnated bone. Note the spaces with jagged margins (A). They contain many fragments of degenerating trabeculæ which tend to end in débris and fibrous tissue. Cells are scanty or absent. There are no giant cells. The presence of vessels is doubtful. (× 40.)

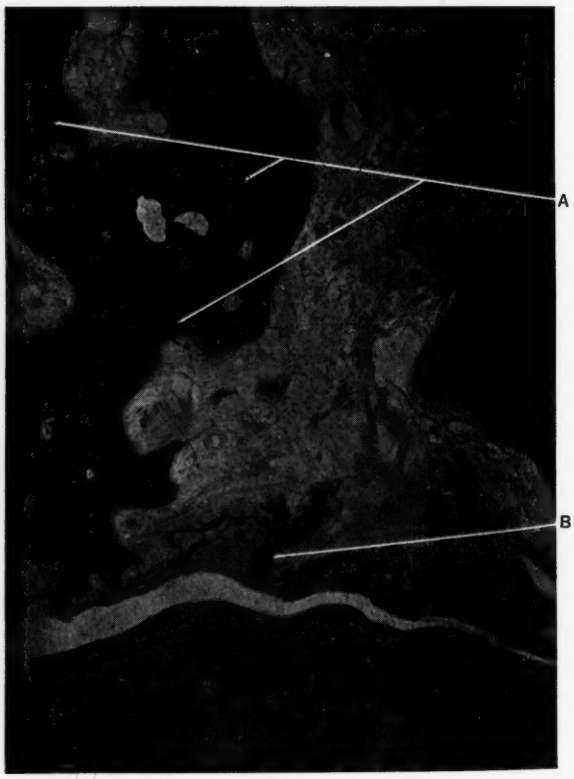

FIG. 241.—Massive fibrous degeneration of bone in Charcot's disease. Section from a femur—Charcot's knee. A, Sclerosed trabecula showing several areas of central degeneration. B, A mass of fibro-connective tissue containing, near the surface, relics of trabeculæ undergoing fibrous resolution — also connective - tissue cells, vessels, and inflammatory cells. It extends deeply between two degenerating sclerosed trabeculæ, and is covered by a thick layer of dense fibrous tissue, containing many connective-tissue cells and some vessels.

(*Specimen* S.C. 88—1.) (× 50.)

Several microscopical sections, however, show osteophytes. Three are almost certainly capsular osteophytes, which have fused with the articular margin (S.C. 85; S.C. 88—1; S.C. 89—1). Another is of doubtful nature, and the fifth—a small marginal osteophyte in an early case—has no doubt developed in the usual way. It shows no sign of recent growth, the medullary spaces are of good size and regular, the trabeculæ are well formed, and the marrow is fatty; but early signs of degeneration have made their appearance in the joint, and the bony surface of the osteophyte shows definite evidence of fibrous degeneration (S.C. 93—2). Evidently the degenerative process has attacked an osteophyte that had settled down to permanency before Charcot's disease developed in the joint. These instances may be contrasted with another specimen (S.C. 22), which is a pretty example of marginal lipping of femur, tibia, and patella with epiarticular osteophytes of the femoral condyles. The patient was a woman, aged 63, who had had rheumatic fever at the ages of 12 and 24, and had suffered from tabes for many years. The knee was swollen and contained fluid and was regarded as a 'typical Charcot's knee'. The left leg had been amputated ten years before for a useless swollen knee, also diagnosed as a Charcot's joint. The specimen, however, has been catalogued as 'osteo-arthritis in a tabetic', for there is no sign pointing to Charcot's disease, either macro- or microscopically.

These facts are opposed to the idea that articular osteophytes are common in Charcot's joints; and it is difficult to avoid the conclusion that the osteophytes usually met with in such cases are capsular formations. Capsular osteophytes, when they are situated near an articular margin, may become attached to the bones; in some cases owing to their increase in size, and in others by ossification in the connecting fibrous tissue of the capsule.

Marginal osteophytes of the ordinary type, met with in definite neuropathic joints, are almost certainly of pre-Charcot development. They will probably show degenerative changes resulting from the tabetic affection.

The origin of capsular osteophytes is of more than ordinary interest, but to avoid repetition it is considered with sclerosis and eburnation, to which it is closely related.

III. SCLEROSIS AND EBURNATION IN CHARCOT'S JOINTS.

Some sclerosis of the joint ends of the bones is not uncommon in advanced Charcot's disease, but eburnation is rare. The Strangeways Collection contains two remarkable macerated knee-joints, in which extensive polished ivory-like surfaces are present on several bones (S.C. 88; S.C. 92). It can hardly be conceived that bone which is the subject of degeneration and fibrous resolution can undergo sclerosis and eburnation any more than that it can develop osteophytes; but there is no doubt that the articular ends of the bones entering into a Charcot's joint, in which destruction has been extensive, may present eburnated surfaces; and that the dense bone may be of considerable thickness. How is this to be explained?

When cartilage or bone is the subject of fibrous degeneration, it is the matrix that is destroyed; and the fibrous tissue that replaces it is derived

from the cells present in the disappearing tissues. The cells are more viable than the matrix. In most cases they proliferate and give either a very cellular or a sparsely cellular character to the fibrous tissue. In some instances the degenerative spaces, and also some of the adjacent medullary spaces, may be so crowded with cells that the fibrous tissue supporting them

FIG. 242.—Articular surface of a femur from a Charcot's knee, showing dense eburnation. (*Specimen* S.C. 88.)

is difficult to detect; in others, cells may be numerous only along the edges of the bone. In the latter case, presumably, proliferation is not so active, or the cells tend to atrophy and disappear, owing to their feeble vitality. In still other cases atrophy overtakes the cells in their cartilaginous capsules or bone cavities, which then appear as empty spaces, whilst the fibrous tissue may be noticeable for its acellular character.

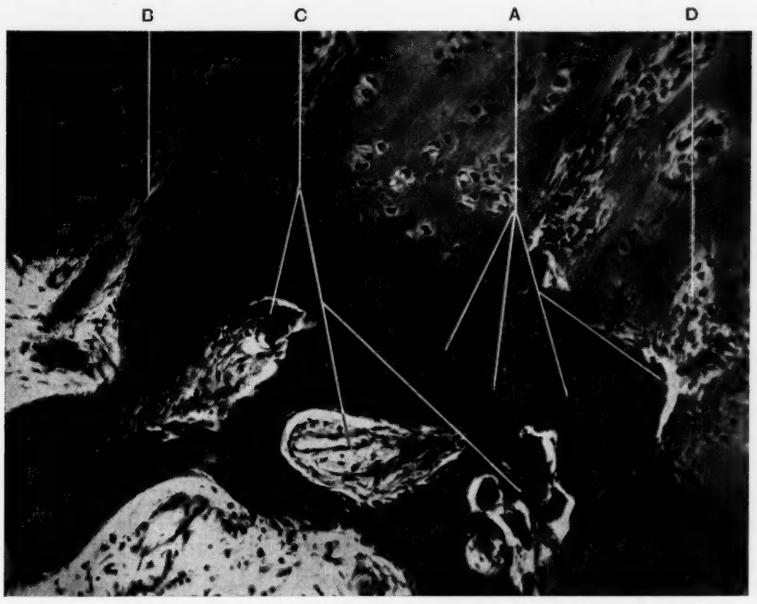

FIG. 243.—Charcot's elbow (right). Section from the head of the radius of Emily K. A, Bone degeneration in subchondroid bone layer—preparatory to formation of spaces; B, The same as A, but fibrous tissue is shown well; C, Formed spaces, two showing giant cells; D, Space forming at the expense of the calcified cartilage and bone. (*Specimen* S.C. 91—1.) (× 110.)

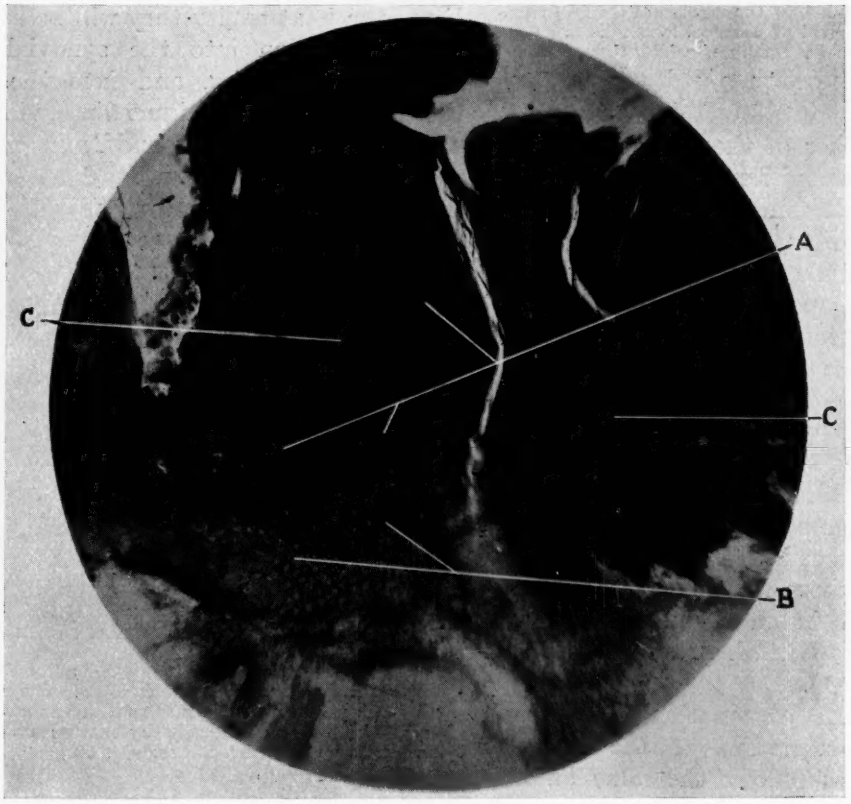

FIG. 244.—Charcot's disease. Section showing degeneration of sclerosed bone from the same knee as *Fig.* 239. A, Spaces full of fibrous tissue showing spaces containing shrivelled cells or their débris; B, Part which suggests fibrocartilage, but the cells are degenerate and the spaces enlarged and in most cases without definite contents; C, Early stage of bone degeneration. (*Specimen* S.C. 85.) (× 75.)

The causes of the degenerative changes in the articular bone-ends in Charcot's joints, are, in all probability, the same as in rheumatoid arthritis, and, as in that affection, the changes may be accompanied by more or less evidence of inflammation or even by necrosis.

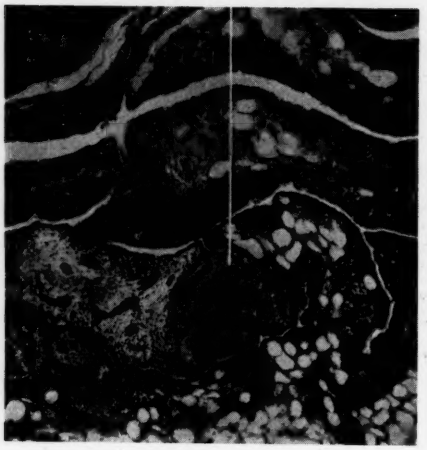

FIG. 245.—Charcot's knee (left). The section is from the capsule and shows a nodule of hyaline cartilage in an area crowded with connective-tissue cells and containing many vessels. Six giant cells are applied to the margin of the cartilage, and several of the cartilage cells are surrounded by a ring of calcification. From an elderly man. (*Specimen* C.S. 88—2.) (× 50.)

The cells resulting from fibrous degeneration of bone and cartilage no doubt possess different degrees of vitality in different cases, but their subsequent behaviour may be largely determined by the kind of cell from which they have originated. Some have developed from the cells of the connective-tissue basis of the cartilage or bone, and others have been evolved from actual cartilage and bone cells. Both may be expected to inherit ancestral proclivities.

Many of these cells must be set free by the rapid disintegration of the articular surfaces and find their way into the joint. From the joint cavity they will be carried by the effusion into the synovial and capsular lymphatics, and into those of the structures in the immediate vicinity. In either of these situations they may be held up (*Toxic and Inflammatory Diseases of Bone*, p. 112), and under suitable conditions may proliferate, and originate new ossific or chondro-ossific formations (capsular and extra-capsular osteophytes), as well as fibrous tissue. It is fair to suppose that the hypertrophic form of the disorder, which is accompanied by numerous capsular osteophytes, is associated with the cases in which the degenerative process is accompanied by a free proliferation of cells, and the atrophic form with those in which the cells disappear in an early stage of the process (*R.C.S. Museum Specimen* 4572—1).

FIG. 246.—Charcot's knee (left). Section through a bony nodule in the capsule from the same knee as *Fig.* 245. The nodule contains cancellous spaces and is growing peripherally by metaplasia of the fibrous tissue in which it lies. (× 50.)

The explanation of sclerosis and eburnation of the articular surfaces follows the same lines as that of the formation of capsular osteophytes. From the nature of the pathological change in the cartilage and bone, it follows that the eroded

articular ends of the bones tend to become covered by an increasing layer of fibrous tissue, in which many of the cells freed from the structures which it replaces, are entangled. The fibrous covering may be excoriated in places

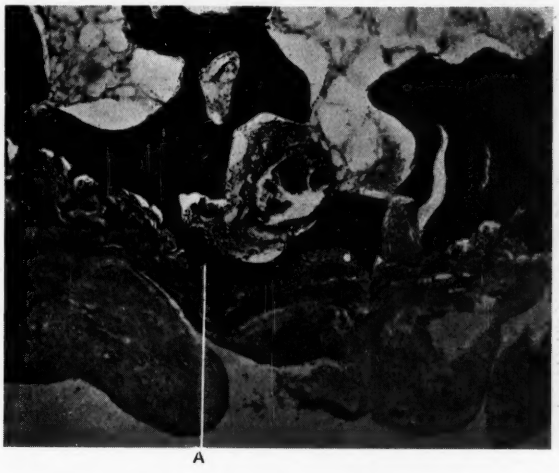

FIG. 247.—Charcot's elbow (left). Section from a deformed and deflected internal condyle. From Emily K., age 62. A general view of the breaking-up articular bony surface; many giant cells. The fibrous covering is composed of dense fine fibrous tissue with many connective-tissue cells, especially in the vicinity of the bone. A, Degenerating area.
(*Specimen* S.C. 91.) (× 50.)

by the rubbing of the bones, but gradually it becomes consolidated, and more or less permanent. The two cases of marked eburnation which have been mentioned show, however, that it is possible for bone to develop in this layer,

FIG. 248.—Sclerosis and eburnation in Charcot's disease (*see Fig.* 250). The section—from a femur of an old man—shows the gradual transition from atrophic cancellous tissue to marked sclerosis and eburnation, with degenerative changes supervening on the surface. The condition bears a considerable resemblance to the similar process in osteo-arthritis. Nowhere is the widespread degeneration of bone, so characteristic of Charcot's disease, to be seen. The sclerosis is advancing by metaplasia of the fibrocellular tissue which occupies the medullary spaces. Where the surface structure is degenerating there are many giant cells among the fibrocellular results, applied along the edges of the disintegrating bone. There are numerous inflammatory cells throughout the section. A, Dense bone, pointing to where a capsular osteophyte has merged with the new articular surface.
(*Specimen* S.C. 88—1.) (× 6.)

and even to go on to sclerosis and the production of a new bony articular surface. A section through the latter in S.C. 88—1 shows that there is a gradual transition from atrophic but otherwise normal cancellous tissue to

dense sclerosis and eburnation, the medullary spaces gradually becoming smaller, and being filled by granulation tissue in which ossification advances by metaplasia.

It is not easy to understand why degeneration should give place to constructive development. In the case of the *sclerosed articular surface* it may be suggested that the fibrous tissue, which replaces the cartilage and bone, is practically scar tissue, and consequently is not supplied with nerves. In an environment no longer dependent on innervation for its stability, the cells find sufficient nourishment to maintain life, and to enable them to develop according to their inherited capabilities. Eventually this results in bone formation, which may go on to sclerosis. The practically normal state of the bone below the sclerosed surface is probably explained by the protection afforded by the new, thick, osseous articular surface. This would save the underlying old bone from the constant friction and slight traumatisms which would be one of the factors tending to promote degeneration.

A similar explanation may be advanced for *capsular osteophytes*. The thick capsule of a Charcot's joint is composed largely of fibrous tissue of inflammatory origin, which has replaced the atrophied original capsule. This, again, is of the nature of scar tissue, and the emancipated cells, brought to rest in it, find a soil in which they can develop their hereditary tendencies. The extravagance and irregularity of the ossification associated with the formation of capsular osteophytes is possibly connected with the loss of the trophic function.

The *absence or rarity of osteophytes in cases of rheumatoid arthritis* which have not passed through a preliminary hypertrophic (osteo-arthritic) stage raises a point of some interest. The Strangeways Collection affords microscopical evidence that small ossific nodules may be present in the chronically inflamed capsule in that affection, but they are rare and inconspicuous.

On the other hand, in osteo-arthritis capsular and synovial osteophytes are a definite feature, and articular surfaces may in some instances be enlarged by fusion of such osteophytes with marginal ones; but they are neither so numerous nor so widespread as in Charcot's disease. The origin of the capsular osteophyte is probably the same in all three conditions, but why are they numerous in osteo-arthritis, practically absent in rheumatoid arthritis, and extravagantly large and widely distributed in Charcot's disease?

Probably several factors are concerned. Free movement of an affected joint is undoubtedly one of the most important. In osteo-arthritis there is slow destruction of cartilage and slower destruction of bone, beginning on the surface. There is considerable effusion into the joint in many cases, into which liberated cells are discharged; and the joint movements are fairly free, even though painful, until checked by muscular spasm or osteophytic locking. In rheumatoid arthritis the degenerative changes begin, and are specially marked, at the junction of the cartilage with the bone; considerable destruction may be limited to this region for some time, and the passage of cells into the joint cavity, at any rate in any considerable quantity, may be prevented by a covering of articular cartilage, which, in most instances, is not quite normal in structure. The amount of effusion is usually moderate and often negligible. The capsule and ligaments are frequently thickened or atrophic and as a rule contracted. The joint movements are painful from

the start, soon become restricted, and finally abolished. The joint is then fixed, and flexed, and adhesions often form between the opposed surfaces.

In Charcot's disease, on the other hand, the destructive changes involve the superficial parts of the articular surface from the beginning, and quickly spread widely and deeply. Large quantities of cells must be set free into the joints. Effusion is copious and often excessive. The joint movements are increased in range, and irregular; being painless, the patient is not induced to control them, and their jolting character must be productive of many trivial injuries which will maintain the effusion and foster inflammatory changes in the capsule.

Evidently the conditions obtaining in the three affections are highly favourable to dissemination of the cells in Charcot's disease, favourable in osteo-arthritis, and the reverse of favourable in rheumatoid arthritis.

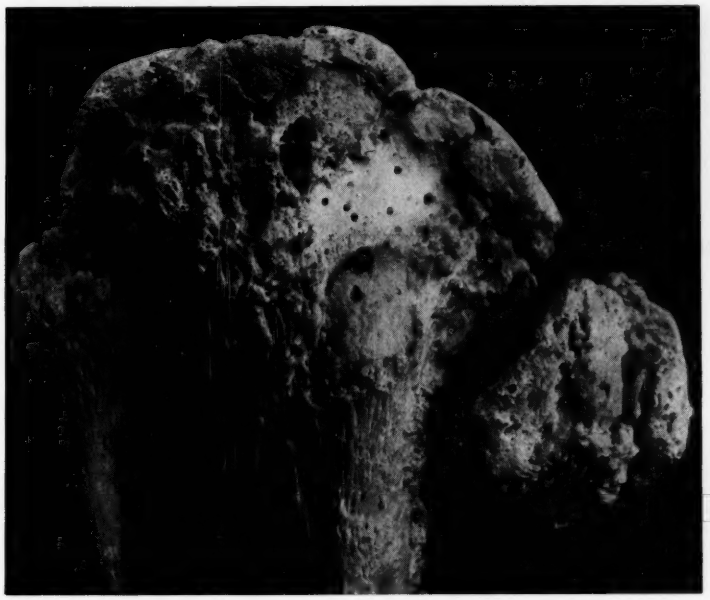

FIG. 249.—The tibia from the same knee as *Fig.* 242, showing the rolled everted edge probably caused by pressure upon softened and rarefied bone. The posterior two-thirds of the upper end of the tibia have been crushed down for some distance so that the part of the upper end seen in the photograph is little more than ½ to 1 in. thick (*see Fig.* 250—the opposite knee). (*Specimen* S.C. 88.)

Spurious osteophytic lipping—an appearance, which I believe is peculiar to Charcot's disease—may easily be mistaken for the lipping caused by marginal osteophytes. It is present in a knee (S.C. 88) in which the lower end of the femur is received into a deep cup formed by the anterior part of the head of the tibia, and its crushed down posterior portion blended with numerous capsular osteophytes. The front edge of this cup is thick, rolled, and everted—a condition that has evidently been produced by pressure upon the softened and degenerated bony margin through the play of the femoral condyles within the articulating cavity.

IV. FRACTURES INTO JOINTS.

The influence of the widespread degeneration and softening, and of the intermittent and irregular pressures to which the ends of the bones are subjected, may be far-reaching. It is obvious that the shape of a softened bone may not only be considerably altered, but a bone-end may even be enlarged and expanded owing to the way in which pressure is applied. An important complication of a Charcot's joint, induced by the bony degeneration, is a fracture communicating with the joint. Such fractures are of common occurrence and are usually overlooked. Owing to the absence of pain a patient may continue to use a limb or a joint which is the seat of such a fracture, and even be unaware that anything unusual has happened. The line of fracture in these cases is more or less obliquely vertical, and a considerable fragment is apt to be separated. The fragments may be held in fair apposition by periosteum and by muscular attachments, and firm bony union may result (S.C. 91—1); or they may be pushed apart by the action or shape of the opposed articulating bone; or, again, the fragment may be crushed and lose its natural shape, or be displaced. It is possible that such a sliding, crushing fracture is of slow and gradual production. Thus the remarkable specimen S.C. 88—1 can only be explained by the slipping downwards and backwards of the posterior three-fourths of the head of the tibia, leaving a thin anterior portion to represent the bone. The chief part of the upper end has been crushed obliquely downwards, and has amalgamated with capsular osteophytes to support and contain the lower end of the femur. The condition is symmetrical, yet there is no history of a fracture,

FIG. 250.—A Charcot's knee, showing a cup-shaped articulating surface which contains the lower end of the femur—see description in the text. From an old man. *Figs.* 241, 242, 248, 249 are from the same patient. (*Specimen* S.C. 88—1.)

and it is specially mentioned that the man was able to get about, even up- and down-stairs, till his death (personal communication) (*Fig.* 250).

In another specimen (S.C. 91) the external condyle of a humerus has been separated, and is incorporated in the capsule more than an inch away from the humerus. Microscopical examination shows it to be a part of the original bone, and not a capsular osteophyte.

SUMMARY OF THE ARGUMENT.

The different rheumatoid conditions result from the action and interaction of certain important factors.

1. The circulation of toxins is probably the essential and underlying factor in all four.

2. The measure of the vitality of the joint tissues is shown by their resistance to the toxic influence, and is the deciding factor in the evolution of osteo-arthritis and rheumatoid arthritis.

3. A metabolic factor is of decisive importance in gout ; and

4. A nervous factor in Charcot's disease, but in both these conditions toxins and tissue vitality take their full share.

I take the opportunity to acknowledge my obligations to those who, often at no little trouble to themselves, tried to get information for me that I wanted. Chief amongst them are the Medical Superintendents of those hospitals or infirmaries from which much of the Collection was drawn. Especially good to me were Drs. Baly and Stebbing. The Lambeth Hospital records are so good that I hardly ever failed to get the information asked for almost by return of post ; and I troubled them often.

The relations of any worker at the College of Surgeons with its Conservator cannot fail to be happy, and in the years spent upon the investigation of the Strangeways Collection he was not only helpful and encouraging on many occasions, but, at the end, a most useful and appreciated critic. The debt I owe to Mr. S. T. P. Laurance is a special one ; to him I took my difficulties on microscopical problems and talked over many points I found it hard to explain. My association with him will always be a very pleasant memory. Dr. C. F. Beadles, the Director of the Cambridge Research Hospital, the late Messrs. Wherry, of Cambridge, and Wagstaffe, of Oxford, and the Rev. C. T. Allan, have all helped me at one time or another. The work has called for continuous help from Miss Glasscock in the cutting of sections from the old specimens, and for patience, industry, and skill from Mr. J. Finnerty, junr., in the dissection and preparation of the specimens. To them, as well as to the others, I tender my grateful thanks. Lastly, I have been again fortunate in the help of my old friend Dr. G. H. Rodman. His skill and experience add enormously to any scientific value to which this article may aspire, and I am greatly indebted to him.

PRE- AND POST-OPERATIVE TREATMENT OF GALL-BLADDER DISEASE.*

By ARTHUR F. HURST,

SENIOR PHYSICIAN TO GUY'S HOSPITAL, LONDON.

In recent years it has become a matter of routine in every case of renal disease in which the question of operation is being considered to examine a specimen of urine obtained from the affected kidney by a ureteral catheter and to make a pyelogram. It is a curious fact that of the corresponding examinations required for the investigation of disorders of the gall-bladder only the second is in common use. This is in spite of the fact that, as we have learnt from an experience of over 300 cases in which both methods have been carried out, examination of a specimen of bile obtained with due precautions through a duodenal tube after injection of magnesium sulphate gives information of great value in many cases in which a cholecystogram appears to be perfectly normal or in which its interpretation is too uncertain to be of much use.

The presence of active cholecystitis can be inferred from the discovery of excess of mucus with epithelial débris and sometimes leucocytes. The infecting organism is generally found to be *B. coli*, but occasionally a streptococcus or staphylococcus or *B. typhosus* is isolated. The presence of pigment granules is confirmatory evidence of infection, and cholesterol crystals in the bile indicate that a cholesterol stone is probably present, even if the evidence obtained by cholecystography is completely negative. Lastly, my colleague, Dr. F. A. Knott, has shown that the discovery of a yellow lipoid material is almost conclusive evidence pointing to the presence of a 'strawberry gall-bladder', a point of much help in diagnosis, as this definitely pathological condition is generally associated with a perfectly normal cholecystogram. I should like in passing to add that the value of cholecystography would be much increased if the radiologist would always palpate the visualized gall-bladder, as tenderness localized to the organ is strong evidence in favour of a pathological condition being present even if the shadow appears to be normal. This is of considerable importance, as tenderness found on ordinary palpation in the supposed position of the gall-bladder cannot be assumed to be due to the gall-bladder, which is often displaced, and, on the other hand, a tender gall-bladder in an unusual situation may easily be missed.

The routine investigation of a supposed case of gall-bladder disease should include a test-meal, as achlorhydria is present in 25 per cent of cases of cholecystitis and nearly 50 per cent of gall-stones. Neglect to treat the gastritis, which is generally the cause of the achlorhydria, is likely to lead to

* The substance of this paper formed the subject of a short address given to members of the Association of British Surgeons visiting Guy's Hospital on May 5, 1932.

disappointment with the result of the operation, as some of the symptoms supposed to be due to cholecystitis are often the result of the associated gastritis and therefore persist after cholecystectomy.

Pre-operative Treatment.—Many cases of cholecystitis can be cured by means of biliary antiseptics and the stimulation of biliary drainage. I was interested to hear Lord Moynihan state that he knew of no new treatment started within the last few years which had so greatly reduced the necessity for surgical interference in any individual disease as the massive doses of hexamine had done in the case of gall-bladder disease. Many cases which still require operation can be brought to a condition of much greater safety by preliminary treatment of this kind, and I am convinced that a fortnight's medical treatment should invariably precede any operation for chronic disease of the bile-passages. Even in acute and subacute cases the temperature often falls rapidly and the patient's general condition improves very greatly after a few days of treatment with hexamine. With obese patients a longer period of preparation is desirable, both with the object of making the operation less difficult and improving the condition of the patient's heart. It is not difficult by suitable diet to make such a patient lose a stone and a half in weight in a month, during which biliary antisepsis and drainage can also be employed.

It has been known for many years that hexamine is excreted by the bile, but Knott was the first to prove definitely that it acts as a biliary antiseptic in spite of the bile being alkaline, although in the bladder it is inactive unless formalin is set free by the acid urine. Thus pure bile obtained through a duodenal tube has no antiseptic action, but if large doses of hexamine are given, the bile, which can be shown to contain unaltered hexamine, is strongly antiseptic, and any bacteria which were present in it before the treatment was begun disappear. Formerly the maximum dose of hexamine which could be given with safety was limited by the supposed necessity of keeping the urine acid. But hexamine is just as efficient a biliary antiseptic when sufficient alkali is given to make the urine permanently alkaline; under such conditions no formalin is set free and enormous doses can be administered without causing any bladder irritation. For over ten years I have been giving with excellent results 100 gr. of hexamine with 60 gr. of sodium bicarbonate and 60 gr. of sodium citrate after breakfast, after tea, and after a glass of milk or water last thing at night.

At the same time biliary drainage can be promoted in cases uncomplicated by stones by giving on an empty stomach the largest dose of magnesium sulphate which the patient can take without getting diarrhœa. A tablespoonful of olive oil three times a day before meals has the same effect as magnesium sulphate in causing the bile-passages to contract and Oddi's sphincter to relax.

Post-operative Treatment.—The day after operation the hexamine treatment should be recommenced and should be continued for two or three weeks, or still longer if there is reason to believe that the gall-bladder infection was only part of a general infection of the bile-passages. The promotion of biliary drainage by Epsom salts is of even greater importance after than before the operation, and it should be used to the exclusion of all other aperients. Its

continued use after the operation is likely to prevent the symptoms which not uncommonly occur after cholecystectomy for gall-stones and which suggest that a stone has been overlooked in the common bile-duct. I believe that they are generally due to the disturbance in the neuromuscular mechanism of the biliary passages which follows removal of the gall-bladder and which gives rise to achalasia or less frequently spasm of Oddi's sphincter.

If achlorhydria is present, this can often be cured by treating the gastritis by lavage every morning with dilute hydrogen peroxide (1 drachm to the pint). If, however, this cannot be carried out, or if the achlorhydria proves to be due to irremediable achylia, 1 to 2 drachms of dilute hydrochloric acid should be given in 5 to 10 oz. of water with orange juice and sugar as a beverage three times a day.

Lastly, it is necessary to say a few words about diet. It appears to have become customary to give patients definite instructions about restricting their cholesterol intake after operations for gall-stones. Thus a patient of mine whose gall-bladder was removed at the end of 1930 for gall-stones was advised by the surgeon to take a cholesterol-free diet, with the result that in the following six months she became very weak and lost two and a half stones in weight. She improved rapidly on being given a full diet. Such restrictions are never necessary. It is doubtful whether they have any material effect on the percentage of cholesterol in the blood or bile, and moreover when the gall-bladder has been removed, there is no place in which sufficient concentration of the bile can take place for new cholesterol stones to form. Stones found in the ducts after cholecystectomy were there before; they never form after the operation. The only dieting which may be necessary is for associated obesity or gastritis, and no restriction of cholesterol is required.

RECONSTRUCTIVE ŒSOPHAGOPLASTY:

WITH NOTES OF A SUCCESSFUL CASE.

By H. H. SAMPSON, O.B.E., M.C.,
SURGEON, GENERAL HOSPITAL, BIRMINGHAM.

The restoration of deglutition after destruction of the thoracic œsophagus is a problem which has exercised the ingenuity of surgeons for many years. While the commonest cause of such destruction is undoubtedly malignant disease, its radical surgical treatment is unfortunately so difficult and has yielded such meagre success that the problem of restoration of deglutition has hardly arisen up to the present time. That earlier diagnosis combined with increased familiarity with the surgery of the posterior mediastinum will eventually bring this disease within the scope of excision seems almost certain.

It is particularly in cases of benign cicatricial stricture that the desirability of restoration is apparent. Such strictures are usually the result of swallowing corrosive fluids (frequently caustic soda) either in error or by design. Unless the stricture is truly impermeable it is probable that gradual dilatation, commencing maybe with the swallowing of a silk thread, will be successful. Plummer[1] and others have reported many cases of successful dilatation in strictures erroneously thought to be complete.

When true impassibility and impermeability are present there can be little hope of relieving the condition by any intra-œsophageal procedure. I believe this stage is indicated when the natural disposal of saliva no longer occurs. So long as anything passes through the stricture one might expect saliva to be retained for a time at any rate in the œsophagus. Progressive hypertrophy and dilatation above the stricture would occur, and, although the contents might be rejected from time to time, the almost continuous expectoration seen in cases of complete stricture would not happen.

Stress must be laid on the importance of establishing the presence of complete occlusion before any reconstruction can be justified, because a review of the literature suggests that in some cases a series of plastic operations which must carry a considerable mortality rate has taken the place of a simpler, safer, but less spectacular form of treatment.

Long before occlusion is complete it is manifest that gastrostomy must have been performed as a life-saving measure. The prospect of a permanent gastrostomy is distasteful in more senses than one. Many of the subjects are young. Apart from the time and trouble entailed in preparing special food and feeding alone and in secret, the unfortunate patient longs for the pleasure and satisfaction of taking food by mouth and swallowing it. The disposal of saliva is another problem not without its embarrassment. It is only when one sees how frequently a receptacle for saliva must be used that one realizes how much is secreted when the œsophagus is obstructed. Roger,

by demonstrating an œsophago-salivary reflex, has explained this increased secretion; but it is probable that in a normal individual there is a continuous flow of saliva and that deglutition occurs much more frequently than is realized. Life must lose much of its attraction to a young man or woman who is bereft of deglutition. Opportunities for social enjoyment are curtailed and there must be a serious handicap to success in any walk of life.

It is not without significance that in all the reported cases of benign fibrous stricture to which I have had access the thoracic portion has been affected. One knows from watching the swallowing of an opaque meal on the X-ray screen that the downward movement is normally retarded or even arrested in the lower half of the œsophagus. It would seem that the rapidity of passage of caustic fluid over the upper half prevents that depth of burning which results in contracting cicatrization lower down. (Mr. C. W. Gordon Bryan has recently informed me that he has a patient with a fibrous stricture of the cervical œsophagus $1\frac{1}{2}$ in. from its commencement.)

The literature of œsophageal surgery reveals that during the present century every possible avenue of approach to the thoracic œsophagus has been attempted. The only record I can find of an intrathoracic operation for benign cicatricial stricture is that of Llobet,[2] of Buenos Aires, in 1900, who performed paravertebral extrapleural mediastinotomy and divided two strictures. The patient died eight days later.

The construction of an extrathoracic conduit which circumvents the stricture and re-establishes continuity between pharynx and stomach has been the aim and ambition of enterprising surgeons since Bircher[3] in 1894 made the first recorded attempt. Both his patients suffered from carcinoma, and died before completion of the work. His method of constructing an ante-thoracic skin tube has proved the most successful, and is to-day used for the main connection from neck to epigastrium. In both cases death supervened before any attempt could be made to anastomose the upper end of the skin tube to the cervical œsophagus.

Wullstein[4] in 1904, working on the cadaver, was the first to suggest that jejunum might be utilized to connect the stomach with a skin tube. Lexer[5] in 1911 published records of a successful case treated by the Wullstein method. He pointed out that the skin tube had no adverse effect on deglutition, thus disproving Bircher's belief that solid food would not pass down a tube devoid of peristalsis.

In 1925 Rovsing[6] described a method similar to that of Bircher. The skin tube was extended downwards to include the gastrostomy without the intervention of any other structure. He published records of two successful cases. Mention must be made of Jianu's[7] method modified later by Rutkowski[8] whereby the greater curvature of the stomach is utilized to act as the connecting channel with the front of the chest. Dengel[9] has recently reported a successful case treated by this method. Three years and three months elapsed between the first operation and final healing. I cannot help feeling that time would have been saved had the skin tube been constructed at the first operation.

It would not serve any useful purpose to enumerate every attempt that has been made to utilize the various portions of the alimentary tract to bridge

the gap between stomach and cervical œsophagus. Many failures have demonstrated the futility of expecting any alimentary viscus to retain its blood-supply when stretched up to the episternal notch. General agreement has been reached that a skin tube is necessary for at least the antesternal region. Whether this be continued downwards to include an ordinary gastrostomy as in Bircher's and Rovsing's methods, or be continued as jejunum or stomach flap is a matter of individual choice. By far the largest number of successes have attended the use of a skin tube with jejunum as the connecting link. It is this method that I used.

I would urge the advisability of constructing the skin tube first, even though the length required may be difficult to forecast. In the absence of saliva and gastric or jejunal secretion it should not be difficult to ensure primary union of the skin plastic. Although the effective anastomosis of the upper and lower ends of the skin tube with œsophagus and jejunum will still remain the most precarious undertaking, it must be manifest that absence of scarring and recent infection will increase the likelihood of success.

CASE REPORT.

V. B., female, aged 27 years.

SUMMARY.—

May, 1927.—Swallowed ammonia. Onset of dysphagia two months later.

September, 1927.—Unable to swallow solids or liquids. Two dilatations through œsophagoscope produced only temporary improvement.

October, 1927.—Gastrostomy.

February, 1928.—Two further attempts at dilatation restored some swallowing of liquids.

April, 1928.—Unable to swallow saliva. Stricture divided by diathermy. Subsequent development of mediastinitis and broncho-œsophageal fistula.

January, 1929.—Three further attempts at dilatation failed.

February, 1929.—Reconstruction of œsophagus commenced. (1) Antesternal skin tube fashioned. (2) Cervical œsophagus anastomosed to upper end of skin tube.

April, 1929.—(3) Jejunal loop inserted between lower end of skin tube and stomach.

June, 1929.—Unsuccessful attempt to close fistula at upper end of jejunum.

September, 1929.—Second attempt to close fistula.

December, 1929.—Final healing.

HISTORY.—The patient was a female, 27 years of age. In May, 1927, after disappointment in a love affair she attempted to commit suicide by drinking ammonia. After treatment at the General Hospital, Birmingham, she was transferred to the Infirmary, where she remained for one month, and apparently made a satisfactory recovery. Dysphagia was first noticed in July of the same year, firstly with solid food. In September she was admitted to the General Hospital under my colleague, Mr. Woodman, as the dysphagia had become almost complete. Œsophagoscopy then revealed a stricture at the level of the 5th dorsal vertebra; free bleeding occurred from active

ulceration. The stricture was entered for 2½ in., but was not traversed; dilatation was found to be impossible. An X-ray at the time demonstrated the stricture. A week later œsophagoscopy was repeated. The upper end of the stricture admitted a No. 10 filiform bougie; active hæmorrhage occurred and further dilatation was not considered safe. Subsequently swallowing was impossible for twenty-four hours. Egg flip was then swallowed with difficulty for two days, when suddenly deglutition became impossible. The weight was now 5 st. 12 lb. Gastrostomy by Senn's method was carried out by my Assistant Surgeon, Mr. Scott Mason. The stomach was reported to be small but with healthy mucous membrane. By February, 1928, the weight had increased to 7 st. 5 lb.

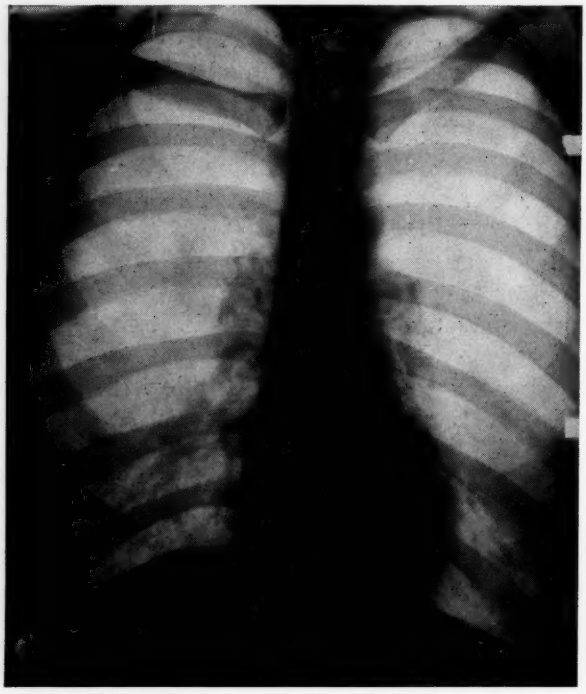

Fig. 251.—Antero-posterior view showing stricture, and barium passing into left bronchus.

In February, 1928, Mr. Woodman again performed œsophagoscopy.

A portion of the stricture was removed for pathological examination. It was found to consist of granulation tissue with abundant cell infiltration. A bougie was passed on that occasion and again a week later. The patient left hospital at the end of February able to swallow a little, but a week later no solid food could be taken, and liquid only with difficulty.

On April 1 she was readmitted to hospital unable to swallow anything, even saliva. Œsophagoscopy by Mr. Woodman demonstrated a stricture 2½ in. long. A bougie was passed and the opening enlarged by diathermy. Some pyrexia followed, but five days later she swallowed a cup of tea. Radiographic examination now showed a broncho-œsophageal fistula just above the stricture (*Fig.* 251).

By December, 1928, she was unable to swallow saliva, and feeding was carried out entirely by the gastrostomy.

Œsophagoscopy by Mr. Adams now showed a stricture 10 in. from the incisor teeth which bled freely and was impassable to any bougie. Three days later a filiform bougie was inserted and dilatation commenced, but deglutition was not restored and it was decided that such treatment would not be successful.

On Feb. 8, 1929, she was transferred to my ward at the General Hospital. Twenty-one months had then elapsed since swallowing the ammonia. She

carried about with her a bottle into which she expectorated every minute or less. The general condition was good. The patient readily consented to submit to any procedure which would restore deglutition.

Operations.—My first operation was done on Feb. 14, 1929. It had for its object the formation of a subcutaneous skin tube to extend from the upper to the lower aperture of the thorax. Two parallel longitudinal incisions 2½ in. apart were made from the suprasternal notch to below the ensiform cartilage. The right-hand edge of the strip of skin was undermined for 1 in., the left-hand edge for ½ in. The two edges were then rolled over and united to each other by interrupted stitches of very fine silk, passing through cuticle only and with the knots lying on the inside of the tube. The suture line was reinforced with fine interrupted catgut sutures. The lateral edges were then undermined and flaps mobilized so that they could be approximated and united with sutures. It was difficult to avoid tension at the upper and lower ends. Unequal undermining of the sides of the strip had succeeded in preventing the two lines of sutures being superimposed. It also brought the skin tube to the left of the mid-line. Satisfactory healing by first intention ensued. (*Fig.* 252.)

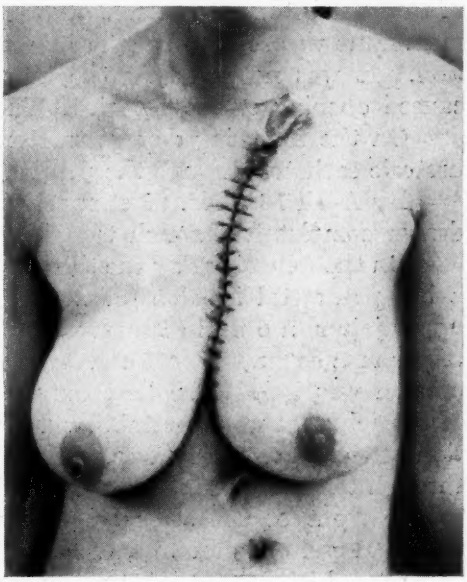

Fig. 252.—Photograph showing completion of first stage of operation. The gastrostomy opening is shown below the lower opening of the skin tube.

Fourteen days later the second stage of the operation was undertaken. It was witnessed by members of the Otolaryngological Section of the Royal Society of Medicine who were then visiting Birmingham.

The cervical œsophagus was exposed through a longitudinal incision extending upwards from the upper end of the skin tube at the inner end of the left clavicle. The recurrent laryngeal nerve was displaced medially. The œsophagus was isolated well into the superior mediastinum and divided as low as possible between cholecystectomy forceps. The lower end was closed and invaginated. After preparation of the edges of the upper end of the skin tube a large rubber catheter was passed up the skin tube and made to enter the upper section of the œsophagus. The mucous membrane of the latter was now sutured to the cuticle of the skin tube around the catheter, using the same fine silk sutures with all knots in the lumen; oversewing with catgut stitches completed the anastomosis. A flap of skin on the left side of the neck was mobilized and swung down to cover the raw surface. A small rubber drainage tube was inserted down to the closed lower section of the œsophagus.

Subsequently there was some leakage of saliva from the suture line, with consequent local wound infection. Attachment of a suction pump to the

lower end of the catheter lying in the skin tube did not abolish the leakage. Both the drainage tube and the catheter were discarded within two weeks of the operation. Saliva was then escaping from the lower end of the skin tube and from a fistula at the anastomosis.

Four weeks after the operation a catheter was passed successfully from the mouth through the anastomosis into the skin tube. Leakage from the fistula diminished gradually. On April 2 a No. 18 stomach tube was passed through the mouth past the junction, and on April 6 the same instrument was pushed completely through the lower end of the skin tube.

On April 10, the stomach having been washed out with sodium bicarbonate solution, a cystoscope was passed through the gastrostomy opening. The main object was to ascertain whether there was sufficient stomach to the left of the gastrostomy to permit anastomosis with jejunum. The gastric mucous membrane was seen to be healthy; there were no signs of scarring or contraction. It was noticed that the introduction of sodium bicarbonate solution caused opening of the pyloric sphincter.

On the following day gastrostomy was repeated; an attempt to pass a ureteric catheter through the cardiac opening of the œsophagus failed.

By April 16 a No. 22 stomach tube could be passed from the mouth and drawn completely through the epigastric opening.

On the following day the third stage of the operation took place. Through a left pararectal incision on the outer side of the gastrostomy a portion of upper jejunum 6 in. in length was selected with a maximum length of mesentery and a separate sheaf of vessels from the posterior abdominal wall. This portion was isolated so that only its vascular supply remained. Continuity of the jejunum was re-established by end-to-end anastomosis and approximation of the mesenteric edges. The isolated jejunal loop was brought upwards through the transverse mesocolon and the anterior layer of the great omentum to the front of the stomach, into which its distal end was sutured. The proximal end of jejunum was brought through the upper angle of the abdominal wound into the subcutaneous layer, where it was united to the lower end of the skin tube by the technique already described. Great care had to be taken to avoid tension on the vascular pedicle. This did not permit easy approximation of the antimesenteric edge of jejunum to the anterior edge of the skin tube. Here some of the sutures tore through the bowel. Undermining of the skin enabled all raw surfaces to be covered in and no drainage was employed.

After nine days of satisfactory progress a drink of water was given. Some leakage occurred through both suture lines. Ten days later a No. 12 stomach tube was passed into the jejunal loop, and on May 21 the same tube was passed into the stomach. By May 30 the patient was drinking fluids freely. The fistulæ were covered by pads and strapping. Leakage was not enough to be troublesome.

In June an unsuccessful attempt was made to close the lower fistula by a small plastic operation. The patient left hospital in July taking all her food by mouth and with the upper fistula healed; pressure by hand prevented leakage from the lower fistula. In September she was re-admitted for another plastic operation to close the fistula. It was only partially successful. Later

the edges were cauterized, and by December healing was complete. The gastrostomy opening closed spontaneously.

Subsequent History.—Shortly after discharge from hospital the patient returned with a lump of meat impacted at the upper skin junction; within an hour it had passed on without treatment; no further difficulty has occurred. She leads a perfectly normal life, eating and swallowing ordinary food (*Fig.* 253). Her weight is now 8 st. 1½ lb. During deglutition the bolus can be seen to descend the skin tube rapidly. It is then carried on into the stomach by visible peristalsis of the jejunal loop. That gravity is not necessary to convey food through the skin tube was demonstrated by the successful swallowing of barium paste when she was lying in an inverted position (*Fig.* 254). The radiographic screen showed that the heavy opaque meal quickly passed into the stomach, its passage being hastened by repeated swallowing of air (*Figs.* 254–256). Dr. Newton, of Moseley, suggested that deglutition was accomplished by air pressure,

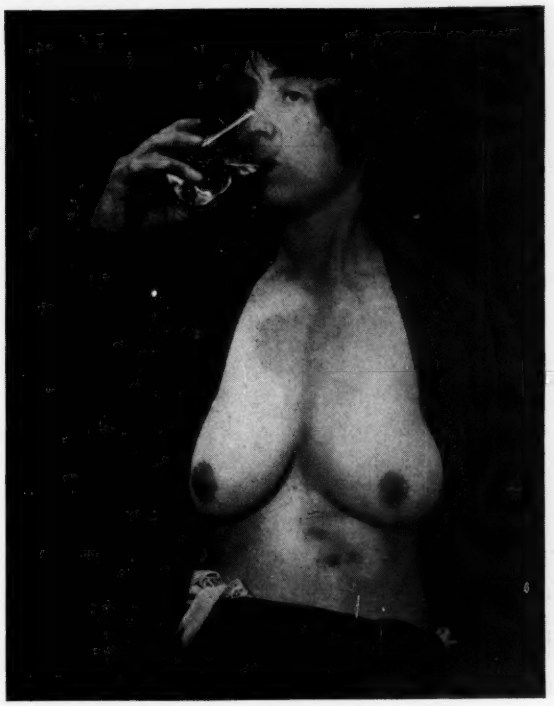

Fig. 253.—Photograph showing patient drinking. Active peristalsis is visible in the subcutaneous loop of jejunum.

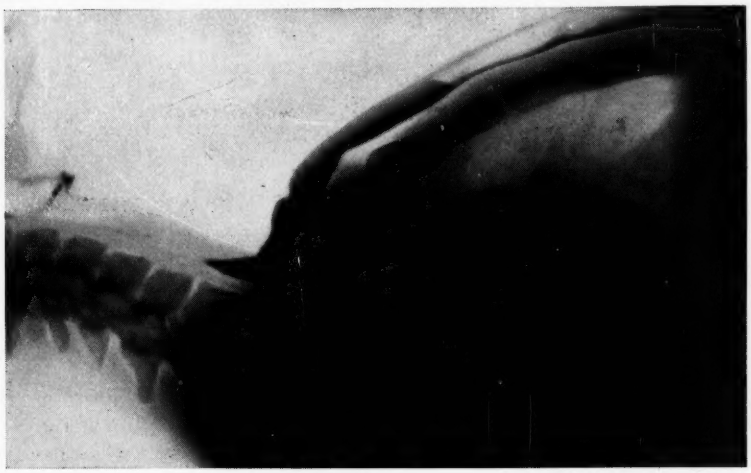

Fig. 254.—Lateral view of new œsophagus with the patient inverted, showing barium passing through upper skin junction and air-bubble in front of sternum.

and this may be the explanation of the rapid descent of food without the aid of œsophageal peristalsis. The patient has not vomited since her first operation. Whether vomiting could occur without reverse peristalsis of the jejunal loop is an interesting speculation.

I desire to record my acknowledgements to Professor Zaaijer, whose successful case at Leyden was my inspiration; to Mr. Woodman, who provided me with the opportunity; to my House Surgeons, especially Mr. Knight and Dr. Taylor, for their valuable help; and to Professor Lockhart for assistance with the illustrations.

FIG. 255.—Lateral view of new œsophagus showing barium passing through lower skin junction and jejunum into the stomach.

FIG. 256.—Antero-posterior view of same state of affairs as in *Fig.* 255.

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BIPARTITE CARPAL NAVICULAR BONE.

By G. I. BOYD,
DEPARTMENT OF ANATOMY, WINNIPEG, CANADA.

Though the occurrence of congenital bipartite navicular bone is referred to in surgical writings,[2, 3, 4, 9] the condition appears to be rarely found and its existence sometimes forgotten altogether.

A divided navicular bone was found in the right carpus of a male dissecting-room subject. The bone was divided about its middle, the line of division passing from the articular surface for the radius, near its lateral

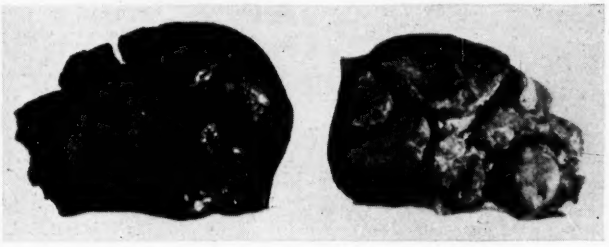

Fig. 257.—Dorsal aspect of dissected carpi. Left and right.

edge, to the middle of the concavity for the head of the os capitatum, so that there was a larger radial portion, distally situated, articulating with the ossa multangula, and a smaller ulnar portion articulating with the os lunatum.

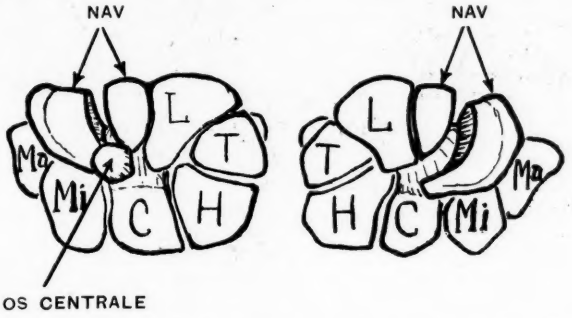

Fig. 258.—Diagram of *Fig.* 257. L, Lunate; T, Triangular; H, Hamate; C, Capitate; Mi, Lesser multangular; Ma, Greater multangular.

This is the usual site of division. Rarely the bone is divided into dorsal and palmar portions.[1, 6] The opposing surfaces of the two portions were clearly defined and covered by a dense layer of eburnated bone without any cartilage apparent to the naked eye. The cavity between them was separated from the cavity of the radiocarpal joint by a ligament uniting their proximal edges.

In the discussion which arose among my colleagues as to whether this was a fracture or a congenital division of the bone, it was suggested that the other carpus be examined. It was found that the latter had been dissected two years previously and preserved in a jar—hence the discoloration evident in the photograph (*Fig.* 257). It presented a precisely similar appearance to the right carpus, with the additional presence of an isolated bony nodule attached to the dorsal aspect of the radial portion of the navicular bone

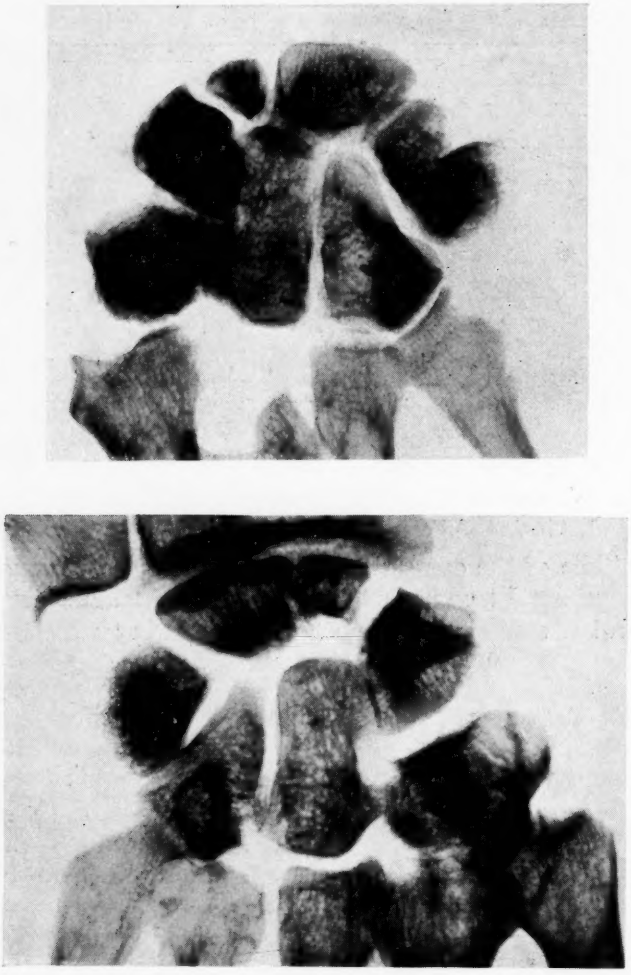

FIG. 259.—Radiogram of left and right carpi, taken after dissection, so that joint spaces are opened up.

where it articulated with the capitate (*Fig.* 258). The radiogram (*Fig.* 259) demonstrates the well-defined adjacent edges characteristic of bipartite navicular bone.

The navicular bone is usually described as ossifying from a single centre about the sixth year. Pfitzner[13] has shown that it is formed of two main elements, a radial and an ulnar, and two accessory bones—the os radiale

externum, which is incorporated in the tuberosity but may be separate,[7] and the os centrale, which may, however, fuse with the capitate or remain as a separate ossicle on the dorsum of the carpus. A double cartilaginous centre has been found by Bardeleben, Thilenius, Rambault, and Renault (cited by Wolff[18]), though other writers have reported negative results.

Dwight[3] has found the bone subdivided at birth. The same author states that in a considerable number of cases, probably 2 per cent, there are signs of an incomplete division, or, more accurately, of an incomplete union of two originally distinct elements. He describes a post-mortem specimen of a divided navicular bone of the right hand in which the opposing surfaces were in perfect apposition and permitted no movement. Microscopical examination confirmed the presence of a cartilaginous coating.

It is admitted that the majority of specimens have a pathological appearance, with little or no intervening cartilage, and polished, eburnated, opposing surfaces, possibly because they are not anatomically adapted to form a good functional joint. This may be accompanied by arthritic changes in the other carpal bones. It may thus be difficult to distinguish a congenital divided navicular bone from an old fracture with non-union and subsequent osteo-arthritis. Indeed, the cases reported by Struthers[15] and Johnson[8] were ascribed to fracture.

The case described by Leboucq, cited by Gruber,[6] was bilateral. In several instances, and in the case now described, a supernumerary carpal bone was present, especially an os centrale.[6, 8, 17] In some cases it might be regarded as abnormal ossification occurring in the dorsal ligaments as a result of faulty mechanics, but in the present instance the position of the bone and its clear demarcation from the surrounding ligaments suggest that it is probably an os centrale.

Most of the reported specimens of congenital bipartite navicular bone have been found in the cadaver. Pfitzner found the condition unilaterally 9 times in 1456 wrists, but it is contended that some of these cases may have been fractures. Gruber[6] examined 3007 navicular bones, of which 4 were bipartite and 1 was tripartite, but in the next 1000 only 1 was divided, and this indefinitely.

Codman,[2] in looking through 1040 X-rays of wrists, did not find any divided scaphoid which was not definitely associated with injury" (except for three cases which he considered to be fractures on account of the marked osteo-arthritic changes present). Köhler[10] has not seen an os naviculare bipartitum in an X-ray, and "agrees with Wolff in supposing that the majority of the cases of naviculare bipartitum are simply pseudo-arthroses following upon fractures."

Schulz[14] records a case of bilateral congenital bipartite navicular bone which was revealed by a radiogram. There was no history of injury, but the patient complained of pain in the wrists which began at the age of fifteen. Mouchet[12] describes a case of congenital bipartite navicular bone in the left wrist of a young man who had developmental shortness of the phalanges. There had never been an injury and the condition was diagnosed by X rays. Faulkner[4] quotes a case with no history of any trauma which Blencke diagnosed by X rays. He himself describes a case of gonorrhœal arthritis occurring in the joint between the two portions of a divided navicular bone.

The condition was bilateral and there was an interval of a few months between the onset of symptoms on the two sides. Radiograms were taken and both sides were subsequently operated on. Naked-eye examination showed irregular surfaces coated with degenerated cartilage.

McKendrick[11] does not mention congenital bipartite navicular, but states, "it is no uncommon thing to find a fractured navicular when X-raying the wrist for conditions other than injury." He ascribes such cases to an old fracture which has been forgotten by the patient, but it is just as likely that they are cases of bipartite navicular. Indeed, McKendrick himself states that it takes considerable violence to cause fracture of the bone. Todd[16] discusses the differential diagnosis between congenital division and fracture of the body of the navicular bone. He points out that the violence necessary to break it is not likely to be forgotten and that the history is definite and characteristic. A recent case of fracture shows unmistakable physical signs which are usually diagnostic of the condition without the help of X rays.

SUMMARY.

There appears to be good evidence for the occasional occurrence of a congenital bipartite navicular bone, probably about once in a thousand times.

It is to be recognized: first, by the absence of a definite history of injury; secondly, by the meagreness of the physical signs; and, thirdly, by the presence in an X-ray picture of a thin well-defined and continuous line produced by the layer of compact bone covering the spongy bone. In the case of a fracture, a broader, less sharply defined line may be expected, which may show irregularities in width. Fourthly, a similar appearance, if present in the other wrist, is strong confirmatory evidence, but the presence of a normal navicular on the other side does not invalidate the diagnosis of congenital origin.

The condition becomes of special importance in assessing claims for compensation on account of an alleged injury.

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EIGHT LETTERS OF JOSEPH (LORD) LISTER TO WILLIAM SHARPEY.

(*Concluded from p.* 164.)

By C. R. RUDOLF.

11, Rutland Street,
Edinburgh,
26 *Oct.* /59.

My dear Dr. Sharpey,

Agnes wrote to you the day before yesterday enclosing my testimonials, and mentioning, I believe, the circumstances which induced me to send in my application to the Home Office. Of that application I received an acknowledgement from a clerk on Sir G. C. Lewis'[19] behalf; and I also know that the Lord Advocate has received his copy of the testimonials: so that I am now fairly in the field. And yet it seems again quite uncertain whether Lawrie has really resigned: at all events, if he has, neither the Glasgow Professors nor the Lord Advocate know anything of the matter. And yet I am well satisfied that I have sent in my application. Benjamin Bell,[20] who is kindly interested on my behalf, has seen the Lord Advocate, who told him that when he was in London (some months ago) Fergusson had pressed him very hard regarding Edwards, but he seemed anxious to have a further conversation with Mr. Bell, and also agreed to act on his suggestion that he (the Lord Advocate) should also refer to Allen Thomson and Douglas Maclagan. The latter is now fully on my side, Spence having retired from the field, and has great influence with Moncreiff, and, withal, is President of the College of Surgeons this year. Benjamin Bell says the Lord Advocate seemed quite open to conviction, and promised to read my testimonials carefully. So things look pretty favorable in that quarter.

Also Sir James Clark[21] called on Mr. Syme to-day, and had a conversation with him on the Glasgow business, and seems quite clear as to the superiority of my claims. I mentioned to Sir James (when I saw him for a minute or two) that I had stated in my letter of application to the Home Secretary that Sir James was willing to act as referee; and he quite approved. Whether or not he may be referred to is, however, quite uncertain, and I rather expect, from Sir James saying that "it would come best in that way", that he will not address the Home Secretary unless he be applied to. I have sent copies of the testimonials to the Glasgow Members of Parliament and also to the Professors, but beyond this I am keeping pretty quiet till the vacancy is distinctly declared. I have gone on talking about my own affairs in a way that would be hardly pardonable did I not know that you are interested in it.

And now my sheet being full, I had better conclude, remaining with kind regards from Agnes to you,

Yours ever most sincerely,
Joseph Lister.

W. Sharpey, M.D., Sec. R.S.

2, Rutland Street,
Edinburgh,
27th March /64.

My dear Dr. Sharpey,

Happening to be here at the time your letter to Mr. Syme arrived, I had the great pleasure of learning that you were the author of the remarks on Medical Education in this week's *Lancet*, and therefore that you approve of the plan of substituting class examinations for final examinations of degrees and licences in the Scotch schools. In Glasgow the Medical Faculty have unanimously agreed to adopt this principle, and the Senators at their last meeting acquiesced in the proposal. So we shall begin to work the new system this session, and I am sure it will prove an *immense* boon to the better class of our students. It will both act as a powerful incentive to work at each subject earnestly while they are engaged in it, and also leave their minds free to engage in the work by taking off from their shoulders the heavy load of the "cramming system". I was at one time afraid that the Assessors would have more work thrown upon them than they could accomplish; but it occurred to me that all that would be necessary would be for each Professor, when he has himself ascertained which of his students have exhibited sufficient attainment, to shew to an Assessor the written answers of these good students only. These will probably not be more than about a quarter or a third of the class, if so many, and thus the Assessor's labour will be greatly lightened. We are accordingly intending to act in this way: and it is a very fortunate thing that, thanks to the advice you gave me (which advice as coming from you was I believe a main means of enabling me to introduce the thing) we in Glasgow have for the last three years given our students class certificates of four different orders of merit. All that will be necessary will therefore be for the Assessors to look over the answers of those whom we think deserving of first and second class certificates, and then it will come to be understood that the receipt of such a certificate by a student exempts him from further examination. I believe that this system will so lighten the students' labour that any reduction of the curriculum will be quite unnecessary. For example, take even Botany. I think it will be anything but a disadvantage to a man to attend a course of Botany in the summer session of his first year, acquiring practical knowledge of plants by the excursions, etc., and learning habits of observation; *provided* that at the end of this course he can feel that the subject need not trouble him further. I concur most cordially in what you say about the essential thing being to get a man to work *really well* at a subject *once*.

The appointment of the Assessors was also a most happy thing for us. Without them we could not have worked this new plan of examination: which in truth will exercise a scarcely less beneficial effect upon the examiners than upon the students. For some of our Professors, I am sorry to say, did the examining duty in a very imperfect slovenly way, so that a student who only deserved a 4th class certificate might get a 1st and vice versa; which not only caused dissatisfaction in times past, but would have proved fatal to the proper working of the new system. Now, however, the Professors will take very good care that they do not send up to the Assessors answers from their

students of which they ought to be ashamed. And indirectly a good effect will also be produced upon our teaching, for it will not do for us to give hobbies too prominent a place or omit other matters of essential moment. I cannot tell you the happiness I feel in looking forward to this plan. I have been deeply impressed with the evil of having the student's mind occupied merely with a sort of unhealthy stuffing of the memory, to the utter neglect, if not destruction of the judgment, which is, I think, the most important faculty for a medical man. I think in Glasgow there has been a peculiar tendency to this: the very diligence of the students seeming often almost mischievous, from the slavish way in which they learnt what they were taught. And yet the poor fellows had no leisure for anything else.

With regard to the practical examination I must say I do not quite agree with you. As to anatomy there is no question of the immense advantage any more than of the practicability of the mode of examining, and Mr. Syme too is far from objecting to it there. But the case of Medicine and Surgery is altogether different. Letting alone the question of the propriety of using patients for the purposes of examination and (especially at a large school such as ours, where we expect 60 candidates to come before us at once on the next occasion) the practical difficulties in the way of carrying out the thing, the grand objection seems to me to be that you *cannot* expect students to be *practitioners* as they are *anatomists*. The best proof of this is that both in Edinburgh and Glasgow it is our *graduates*, and the very best among them, who take the offices of Residents in the Infirmary for the very purpose of doing that with reference to practical Medicine and Surgery which the student in the dissecting room does with reference to practical Anatomy. To be able to diagnose a case and decide at once upon the treatment requires a very high degree of practical attainment. I know for my own part, it was more by good luck than anything else that I was able to make anything like a decent appearance at the practical examination for M.B. at the London University. I never acquired anything *approaching* the sort of style that this examination professes to test till I came to be Dr. Walshe's assistant *after* my graduation. It may be said that it is very desirable to do anything you can to encourage practical work at the cases. But I believe that if the student is relieved from the labour of cramming and at liberty to look at his studies in some other light than merely as means for getting him through his examination, he will see the obvious importance of hospital work, and regard the clinical information he gains as having its own sufficient reward: being in fact the principle stock in trade for his future practice. Thus I confidently expect that under our new system hospital study will flourish with a healthy vigour far superior to that which could be fostered by any Clinical examinations: and I only wish I could clearly see a way in which the same system could be introduced in London.

I did not expect to have troubled you with so lengthy an epistle, which I only hope you will not think presumptuous, but believe that I remain as ever,

Yours with the greatest respect and gratitude,

JOSEPH LISTER.

W. SHARPEY, Esqr., M.D., Sec. R.S.

THE REPORTS OF THE TWO REFEREES (SIR) JAMES PAGET AND JOHN GOODSIR IN THE POSSESSION OF THE ROYAL SOCIETY.

Report on Mr. Lister's Paper on the Early Stages of Inflammation.

This communication consists of an introduction, four sections and two supplements, each of which requires separate notice.

The introduction contains some observations on the early phenomena of inflammation, intended chiefly to prove their similarity in warm and in cold-blooded animals: and an account of the theories of previous writers respecting the stagnation of blood in an inflamed part. Some of the observations, though simple, are I believe novel as well as instructive and should be published. The account of the theories may safely be omitted (pp. 6–10).

The first section "On the aggregation of the Corpuscles of the Blood" seems to me all worthy of publication.

The second "On the Structure and Function of the Blood-vessels" would be improved, I think, by the omission of all the discussion of vital affinities, etc., contained in pp. 22–25. The author appears to have laboured under the influence of recent discussions in Edinburgh, which, in both this and other parts of the paper has led him to give unnecessary extent to comparatively trivial and uncertain things. The rest of this section is valuable and necessary to the understanding of the parts following it.

The third section "on the effects of irritants upon the circulation" deserves publication entirely.

The experiments are simple and instructive—they confirm, with more evidence than has yet been published, the view that the stagnation of blood in the living vessels is not dependent on changes of size of the vessels, but, probably in molecular or vital changes in their walls, or in the tissues external to them.

The fourth section is the most valuable of all—the observations on the pigment cells of the frog's web give the best or only visible proof yet discovered, that in the early stages of inflammation in vascular parts, the tissues external to the blood-vessels are independently and even primarily affected by the stimulus applied. The whole of this section should, I think, be published, but I would suggest that the expressions about "paralysing" the attractive and repulsive, or the concentrating and diffusing, forces in the pigment cells imply much more than is proved and might be advantageously changed.

The concluding remarks, pp. 63–68, which seem to have been written under the influence already referred to, may be safely omitted.

Of the two supplements, the first, "On the influence of the nervous system on the arteries" should be published, if at all as a separate paper. And I should certainly recommend it for publication, but that I think the author could much increase the value, by increasing the precision of his results, if he would continue and extend his observations—. At present he has not attained to much more than the proof that the lower part of the spinal cord in the frog exercises an influence on the blood-vessels in the hind

feet similar to that proved by M. Brown-Sequard and others for the spinal cords of mammalia—. It would be interesting to imitate M. Brown-Sequard's experiments on sections of one lateral half of the cord, and to contrast the states of the vessels in the two webs: and by this and other experiments, I think, the author would discover more precisely than he has yet done, the seat of the nervous centre of the blood vessels. I would suggest, therefore, that instead of publishing this supplement in the Transactions, the communicator of the paper should be requested to recommend the author to extend this part of his enquiries.

The second supplement "On the Pigmentary System of the Frog" should, I think, be published entire, and as a separate paper, preceding the essay on inflammation.

JAMES PAGET.

July 30, 1857.

"Mr. Paget's Report on Mr. Lister's Paper"
July 30, 1857.

THE COMMITTEE OF PAPERS, ROYAL SOCIETY.

GENTLEMEN,

Having been requested by you to give my opinion regarding the eligibility of Mr. Lister's "Paper on the Early Stages of Inflammation" for publication in the Philosophical Transactions, I beg to state that while this paper contains observations and results, which, if verified, will have an important influence on the progress of the subject of which it treats, it would at the same time, in my opinion, as a paper on a pathological subject, be more suitable for the Philosophical Transactions if it had been more condensed, especially in the 'Introduction' and 'Concluding Remarks'.

I am, Gentlemen,
Your obed servt,
JOHN GOODSIR.

University of Edinburgh.
Oct. 4th, 1857.

COMMENTARY.

From the time that Lister as a medical student came under the influence of William Sharpey—the first Professor of Physiology at University College—he became his devoted friend and admirer. Under his tutelage was initiated a love of research that was to lead him from his early inquiries in microscopical science and the processes of inflammation to the culminating labours of his antiseptic treatment. It is on Sharpey's advice and with his introduction to James Syme that Lister arrives in Edinburgh, where the first phase of his career was passed and from which place these letters were written. In this correspondence through a number of years—physiology, the Glasgow election, medical education—everything is submitted with deference to his teacher and friend for whom affection and respect are everywhere apparent.

The first, second, third, and sixth of these letters deal primarily with the subject matter of his paper on "The Early Stages of Inflammation",[22] and its two supplements, which, as Lister was not yet a Fellow, were 'communicated' by Sharpey, their Secretary, to the Royal Society. They are also concerned with the criticisms of (Sir) James Paget and John Goodsir, the two referees chosen to decide on the suitability of the paper for inclusion in the *Philosophical Transactions*. The original MSS. is unfortunately lost, but the reports of the two referees are available and are reprinted above. Alterations in format, with the publication of the two supplements, each as a separate paper,[23] are adopted; but Lister is at issue with his critics on the question of 'vital affinities of the blood' and the physiology of the pigment cell. That these 'vital affinities' did, as he suggests, considerably exercise medical opinion at that time is supported by evidence at home and abroad. Alison[24] approves of the theory, and foreign physiologists, including Haller, Magendie, Kaltenbrunner, et al., contribute their observations to the contemporary press. Lister, however, is more concerned with the objection to his term 'paralysis' in relation to the concentrating and diffusive forces of the pigment cell. Writing to his father he says, "I felt it therefore to be by no means agreeable to have the correctness of the expression 'paralysis' questioned by Paget and Sharpey", supplying him also with a résumé of the experiments with which he sought to convince Sharpey. He successfully met both objections, and his views as originally expressed were ultimately incorporated in the *Philosophical Transactions*.[25]

Letters 4 and 5, dated June 28 and July 7, 1858, were the forerunners of his paper, "Preliminary Account of an Inquiry into the Functions of the Visceral Nerves, with Special Reference to the So-called Inhibitory System",[26] which appeared in the *Proceedings* of the Royal Society as a letter to Dr. Sharpey. In this form submission to referees could be dispensed with. There were others in the field and he was anxious to get his views into print, and but little more than two months were allowed to elapse between his first observations and the completed paper. To-day publication in the *Proceedings* is more sought after, but in the middle of the nineteenth century the *Philosophical Transactions* took precedence, its pages being reserved for original work of exceptional importance. It may, therefore, be conjectured that this paper of Lister's was not considered of sufficient moment to warrant, as he wished, its inclusion.

The concluding months of 1859 see Lister's stay in Edinburgh drawing to a close. In August he writes to his father[27] that Dr. Laurie is resigning his professorship in the University of Glasgow for reasons of health, and discusses his own possible candidature. By Oct. 12, it will be seen, he had definitely entered the field from which he emerged the chosen candidate early in the following year.

In 1864 the question of medical education was much to the fore. In Scotland, Syme and Lister led the way, and the Senates of Edinburgh and Glasgow Universities had already reorganized their system of teaching. In London proposals by Syme and Christison for modification of existing practice on similar lines were under consideration by the Medical Council. A series of articles on medical education appear in the *Lancet*,[28] where the

proposed innovations are critically examined. A note of antagonism by no means unusual in the polemics of the time is discernible in its Editorial wherein London teachers are exhorted to formulate their views before the assembling of the Council, whilst fears are entertained that "both the number and the decision of Scotch, Irish and University Members of the Council will give them a great advantage in enforcing their views." In the last of our letters Lister, least combative of men, deals with Sharpey's contribution to the *Lancet* with his usual serenity and good sense.

The original Lister letters are preserved in the Library of the Royal College of Surgeons.

I should like to acknowledge my indebtedness to Sir Arthur Keith for his kind advice, to Sir Edward Sharpey-Schafer for his note on Miss Colville, to Sir D'Arcy Power for his invaluable help in preparing these letters for the press, and to the Royal Society for permission to print the reports of Sir James Paget and John Goodsir.

NOTES.

[1] George Gabriel Stokes, Secretary of Royal Society from 1854 to 1885.
[2] Sir James Paget, 1814–99. Author of *Surgical Pathology*, 1853.
[3] John Goodsir, 1814–67. Professor of Anatomy at Edinburgh University.
[4] William Benjamin Carpenter, 1813–85. Author of *Principles of General and Comparative Physiology*.
[5] Emil Wilhelm, Ritter Von Brücke, 1819–95. Professor of Physiology at Vienna, 1849.
[6] Wilhelm von Wittich, 1821–82. Director of Physiological Institute at Bonn. Worked with Helmholtz.
[7] Emil Harless, 1820–62. Writes from Erlanger on the influence of gases on the blood, 1846.
[8] Eduard Friedrich Wilhelm Pflüger, 1829–1910. Director of Physiological Institute at Bonn.
[9] Augustus Volney Waller, 1816–71. Assistant of J. L. Budge (*see below*). Professor of Physiology, Birmingham.
[10] Julius Ludwig Budge (Senr.), 1811–88. Published his paper on the movement of the iris at Brunswick, 1855.
[11] Tuffer West. Well-known engraver of wood blocks; the business is still carried on by his successors.
[12] Thomas Wharton Jones, 1808–91. Experimental physiologist. Professor of Ophthalmology at University College.
[13] Miss Colville. Sir Edward Sharpey-Schafer writes: "Miss Colville was Dr. Sharpey's niece and kept house for him for a time at Hampstead, but she fell ill and died, I am not sure when. At any rate, as a result, he gave up housekeeping and went to live in lodgings in Torrington Square. She had a brother in either the Indian or Colonial Medical Service, Major Colville, who was stationed for many years at the Consulate at Baghdad and to whom I believe Sharpey left the greater part of his estate; but he did not long survive to enjoy it. He married after his retirement somewhat late in life. I know very well what a high opinion Lister always had of Sharpey."
[14] Lazzaro Spallanzani, 1727–97. Distinguished naturalist. Author of *Observations on the Circulation*, 1777.
[15] Agnes, elder daughter of James Syme and wife of Lister.
[16] Otto Spiegelberg, 1830–81. Professor of Midwifery at Göttingen.
[17] Georg Meissner, 1829–1905. Professor of Anatomy and Physiology at Basel.
[18] Moritz Schiff, 1823–96. Professor of Physiology at Basel.
[19] Sir George Cornwall Lewis, Bart., Home Secretary, 1859–61.

[20] BENJAMIN BELL, 1749–1860. Surgeon to the Edinburgh Royal Infirmary. Not related to Sir Charles Bell.

[21] SIR JAMES CLARK, 1788–1870. Physician-in-Ordinary to Queen Victoria, Physician to the Duke of Kent.

[22] "On the Early Stages of Inflammation", *Philosophical Transactions*, Part 2 for 1858, p. 645. (Read June 18, 1857.)

[23] "An Enquiry regarding the Parts of the Nervous System which regulate the Contractions of the Arteries", *Philosophical Transactions*, Part 2, for 1858, p. 607. (Read June 18, 1857.) "On the Cutaneous Pigmentary System of the Frog", *Ibid.* p. 627. (Read June 18, 1857.)

[24] ALISON'S *Outline of Pathology and Practice of Medicine*, 1843–4, p. 122. "These facts afford a strong presumption that in all these cases the impressions made on the capillaries and on the blood contained in them, solicit the flow through them on the *principle of a vital attraction of the blood*, rather than of relaxation of the vessels."

[25] JOSEPH, BARON LISTER. *Collected Papers*, pp. 219–21, "Vital Affinities." *Ibid.*, pp. 250–1, "Paralysis of Concentrating and Diffusing Forces."

[26] *Proceedings of the Royal Society of London*, vol. ix, No. 32.

[27] *Lord Lister*, by SIR RICKMAN GODLEE, p. 77.

[28] *Lancet*, 1864, March 12, 19, 26.

EXTENSIVE LOSS OF TIBIAL DIAPHYSIS. TIBIO-FIBULAR GRAFTING.

By G. R. GIRDLESTONE,

HON. SURGEON, WINGFIELD-MORRIS ORTHOPÆDIC HOSPITAL, AND HON. ORTHOPÆDIC SURGEON, RADCLIFFE INFIRMARY, OXFORD.

AND W. B. FOLEY,

ASSISTANT SURGEON, WINGFIELD-MORRIS ORTHOPÆDIC HOSPITAL, AND HON. ASSISTANT ORTHOPÆDIC SURGEON, RADCLIFFE INFIRMARY, OXFORD.

The difficulty, or even impossibility in some cases, of bridging a big gap in the tibia after a partial diaphysectomy for osteomyelitis is well known to all who practise bone surgery, and to meet it a new method of bone-grafting was devised and carried out in the case to be described. When recourse is had to direct grafting from the upper to the lower tibial fragment it means that the graft must lie with several inches of its length in tissues which have been septic, are heavily cicatrized, and poor in vascularity, with the result that only too often the central part of the graft dissolves and disappears. Furthermore, the ends of the tibia are generally pointed, sclerosed, and frequently contain small residual foci which lead to infection of the wound and sequestration of the graft. To obviate these great drawbacks it was decided to make use of the fibula to provide a clean, osteogenetic, and vascular bed for the grafts destined to transmit body weight from upper to lower tibial metaphysis. The operation is applicable whenever part of the upper and lower tibial metaphysis remains; and body weight is still transmitted, as it should be, from the upper tibial articular surface to the lower one, the only difference being that *between* these points the weight is transferred to the fibula. The central portion thus consists of fibula strongly reinforced by grafting, and at each end a flying-buttress graft transmits the weight from tibia to fibula above, and back from fibula to tibia again below. The details of operative technique together with the case notes, diagram, and radiographic appearances are appended.

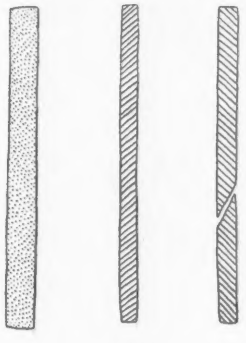


Fig. 260.—A, Delagenière graft, i.e., periosteum with a thin leaf of bone under it taken from almost the whole breadth of subcutaneous surface of tibia. B and C, Albee grafts (cut from area already denuded by the cutting of A); B, Long, to reinforce the shaft of fibula; C, Divided into two grafts to be pegged obliquely from fibula into upper and lower metaphyses of tibia.

Operative Plan.—The idea, in broad outline, was to induce the shaft of the fibula to become as strong as that of the tibia—or at all events, to make it a strong solid bone capable of carrying on by itself the weight-bearing function of the limb. It was proposed to do this by embedding the

grafts obliquely from the fibular shaft into the healthy tibial metaphyses above and below; and to facilitate and accelerate the strengthening of the fibular shaft by reinforcing it with two long grafts, one of the Albee and one of the Delagenière type (*Fig.* 260), these grafts to be laid in a vascular or osteogenetic bed prepared by turning inwards and backwards an osteoperiosteal flap from the front and the inner side of the fibular shaft. It was expected that this strong reinforcement would give the fibular shaft a fair start in its task of making itself competent to carry the weight of the body. A study of the radiograms will show that these hopes have been realized.

CASE HISTORY.

C. C., at that time aged 3 years, was first seen in September, 1927. He was then in the wards of a local general hospital, where he had been for one year, since the onset of acute osteomyelitis of his left tibia in September, 1926. He had had three operations at the hospital, and had a still unhealed sinus over the shaft of his tibia, which was extremely and unduly mobile owing to loss of bone substance. His general health was satisfactory and he was fitted with a weight-bearing caliper and guarding plaster, and allowed to get up.

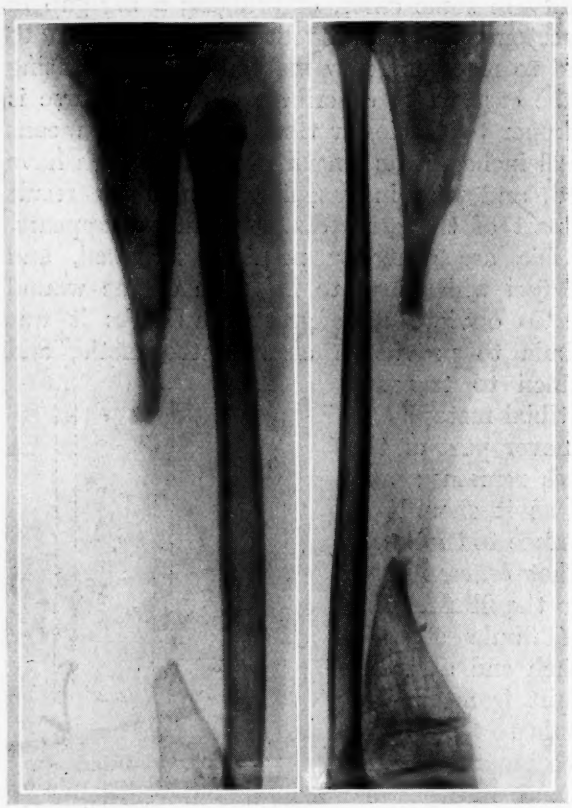


FIG. 261.—X-ray, May 24, 1930, showing the tapered ends with a skin of sclerosed bone, and an oval area in the upper fragment suggestive of a residual abscess.

Four months later (January, 1928) he was admitted to the Wingfield Orthopædic Hospital with a metastatic abscess of the hand. X-rays at this time showed that all the central portion of the tibial shaft had disappeared, and that the ends of the upper and lower fragments were dense and ragged.

This abscess in the hand was opened and drained, and at the same time the tibial sinus was excised and granulation tissue removed from between the bone-ends. It was then left to heal by granulation after packing with vaseline and B.I.P.P.

Two months later (March, 1928) the patient was discharged, with the tibial wound healing well by granulation, in a caliper and plaster with window for dressings.

In June of the following year he was readmitted. The tibial sinus was still not quite healed, so a further operation was done consisting of excision of the sinus track down to and including about an inch of the upper end of the lower tibial fragment. The spiky lower end of the upper tibial fragment was also removed to prepare the way for grafting at a future date. At that time an ordinary tibial graft was contemplated.

A series of metastatic abscesses in various parts of the body now followed, and these were opened and drained, diseased bone and granulation tissue being removed in some cases. The patient was given a course of collosol manganese injections. These abscesses continued up till February, 1930. From this date till June, 1930, he was free from septic manifestations, and was allowed to be up and about in a caliper and plaster, though still an in-patient.

BONE-GRAFTING OPERATION (*Figs.* 262, 263).—This was performed by one of us (G. R. G.) on July 31, 1930. Through a long external incision practically the whole length of the shaft of the fibula was exposed and the periosteum incised and reflected. Tracks were then prepared through the soft parts deep to the anterior tibial muscles, vessels, and nerves, beginning from each end of the exposed fibular shaft and continued as tunnels drilled with a narrow gouge into

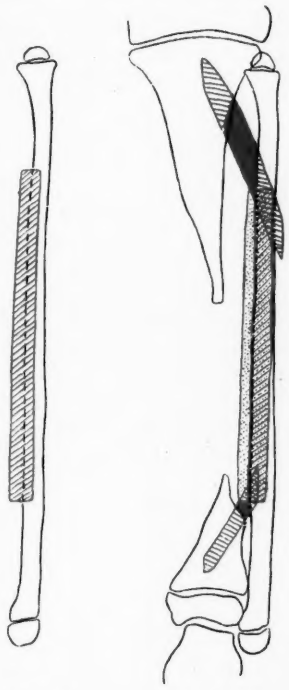


FIG. 262. FIG. 263.

FIG. 262.—Diagram illustrating the reflection of an osteo-periosteal flap from front and inner side of the shaft of fibula.

FIG. 263. — Tracing from X-ray showing the long vertical and oblique peg grafts. In addition (shown stippled) there is an indication of the placing of the Delagenière graft over the Albee (which lies sandwiched between this and the deeper-lying osteoperiosteal flap from the fibula).

FIG. 264.—X-ray, Oct. 2, 1930, two months after the grafting operation.

the healthiest and strongest part of the tibial metaphysis, the upper one obliquely upwards into the upper metaphysis, and the lower one obliquely downwards into the lower metaphysis.

The right tibia was then exposed and four grafts cut as follows : (1) An osteo-periosteal graft about $\frac{3}{4}$ in. broad and sufficiently long to cover the whole length of fibula exposed ; (2) A graft about $\frac{3}{8}$ in. broad, consisting of cortical and medullary bone, of the same length ; (3) Two similar but shorter and rather stouter grafts. The two short grafts, after being pointed slightly at one extremity, were pegged firmly into the tunnels previously drilled in the upper and lower tibial metaphyses.

The free ends of these grafts, after traversing the oblique tracks in the soft parts, ended in contact with the bared shaft of the fibula under the reflected periosteum. The ends were crushed and turned so as to lie in very close apposition to the fibular shaft.

The long bone-graft was then laid along in contact with the shaft of the fibula under the fibular periosteum, and the osteoperiosteal graft was laid over it with its bony surface in contact with the fibular shaft and the three grafts. Both wounds were sutured, and the grafted leg put into a plaster case including knee- and ankle-joints.

Three months later (October, 1930) the plaster was changed. X rays at this time showed all the grafts 'taking' well, and an increase in thickness of the fibular shaft beginning to take place, especially on the anterior surface (*Fig.* 264).

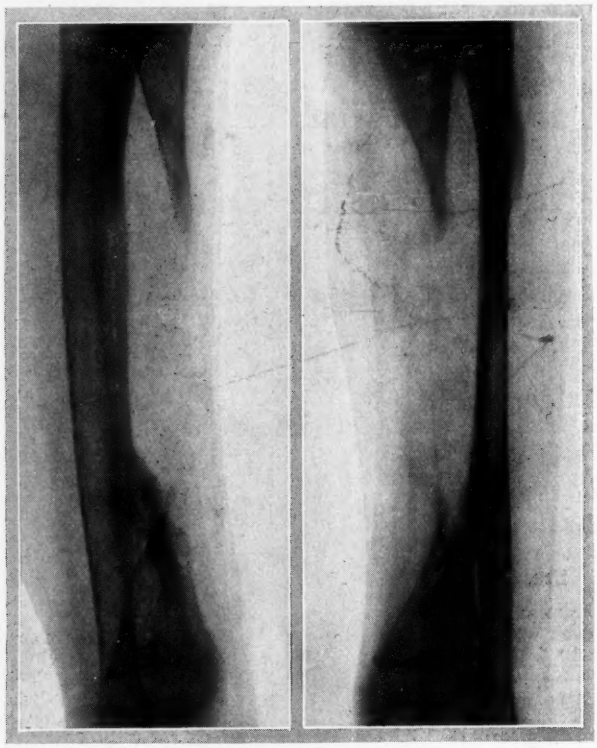


FIG. 265.—X-ray, Dec. 5. 1930, four months after the grafting operation.

A further X-ray (*Fig.* 265) taken two months later (December, 1930) shows a considerable further increase in the solidity of the grafts and resultant thickening of the fibular shaft.

Clinically the leg now felt firm and solid, having lost entirely the old feeling of undue mobility in every direction, due to the gap between the tibial ends. A light modified Delbet plaster was then applied and the patient allowed to walk on this.

The latest X-ray, taken in April, 1931 (*Fig.* 266), shows great hypertrophy of the fibular shaft, which is beginning to resemble in thickness the tibia of a child of this age. The boy is still walking with his Delbet plaster, but this will be removed shortly and walking without it allowed a year after the grafting operation. *Fig.* 267 shows the patient's condition on Nov. 4, 1931.

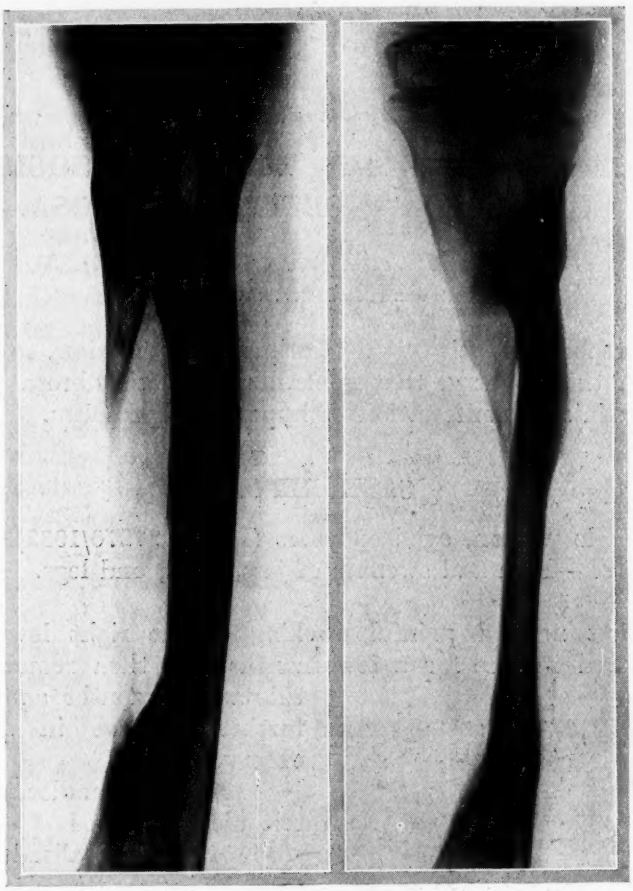


FIG. 266.—X-ray, April 2, 1931, eight months after the operation.

COMMENTS.

It is probable that in subsequent cases bone-grafting by this method would be resorted to even earlier than five months after subsidence of septic manifestation, local or general. Even this interval, however, is short compared to the time that would have to clapse before direct end-to-end grafting of the tibia, through the old inflammatory area, could even be considered.

(N.B.—23.11.32. There is now no shortening, and the boy, in his father's words, "runs about as well as anybody.")

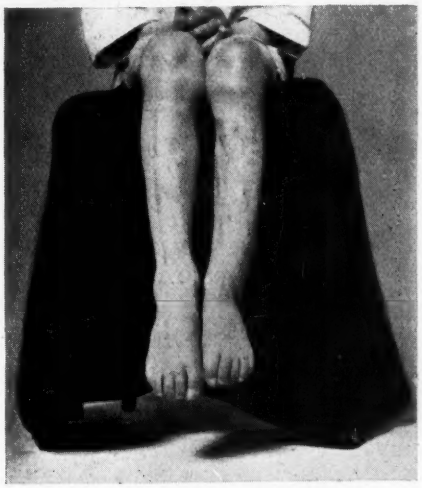


FIG. 267.—Condition of patient Nov. 4, 1931.

A CASE OF PARATHYROID TUMOUR ASSOCIATED WITH GENERALIZED OSTEITIS FIBROSA.

By HENRY COHEN and R. E. KELLY,
ROYAL INFIRMARY, LIVERPOOL.

The case we report below is further testimony to the view, so ably presented by Hunter in this country, that generalized osteitis fibrosa (von Recklinghausen) is a clinical manifestation of hyperparathyroidism.

CASE REPORT.

D. D., single woman, aged 48 years (L.R.I. 27270/1932).

Complaints.—Pain and swelling of jaw, head, and legs.

History.—

1923.—First noticed painful swelling of the right lower jaw, which rapidly increased in size for a few months and then remained stationary till twelve months ago, since when a further increase in size has been observed.

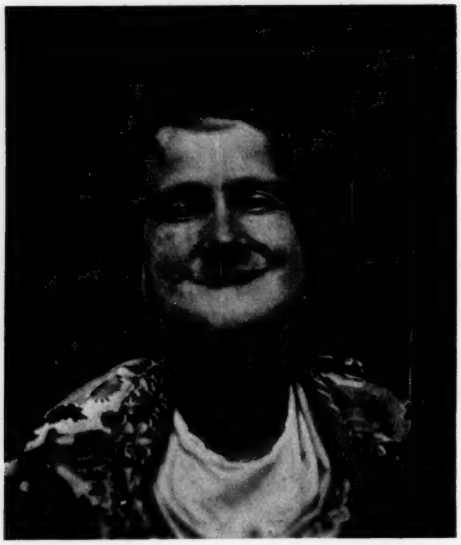


Fig. 268.—Facial appearance and swelling of lower jaw.

1928.—Appendicectomy and ? gastric ulcer removed.

1929.—Generalized headache with gradual increase in size of head.

1930.—First noticed swelling of upper end of right tibia.

July, 1931.—X-ray urinary tract: "Two large and at least two small stones in right kidney. Very suspicious of presence of small stones in left kidney. Abrodil shows hydronephrosis of right kidney."

August, 1931.—Right nephrotomy by Mr. W. R. Williams; four stones removed.

Family History.—No bone disease. Father and mother died of 'senile decay' at 83 and 85 years respectively. Three brothers and four sisters well. One sister has phthisis.

On Examination.—Weight 8 st. 10 lb. Height 5 ft. 4 in. Normal physical development.

Appearance and jaw swelling seen well in *Fig.* 268. Skull normal to palpation and auscultation, but tender on pressure; upper end of right tibia

swollen and tender; no other bone deformity. No anæmia, no clubbing of fingers; sclerotics bluish. No tumour felt in neck.

Alimentary System.—Artificial teeth, tongue clean and moist, no tonsillar or pharyngeal infection, sinuses and antra transilluminate normally. Abdomen: generalized visceroptosis with palpable liver and spleen and right kidney; scars of previous operations well healed. Rectal examination negative.

Cardiovascular System.—Heart normal in size, shape, sounds, position, and rhythm, but systolic bruit audible at all areas, especially mitral. Blood-pressure, 135/70; pulse-rate, 84. No arteriosclerosis, either peripheral or retinal.

Central Nervous System.—No diplopia or visual defect. Fundi, pupils, reflexes, sensation, co-ordination, etc., reveal no evidence of organic nervous disease.

Respiratory System. — Lungs clear.

FIG. 269.—Antero-posterior view of skull.

FIG. 270.—Lateral view of skull.

Genito-urinary System.—No dysuria; no frequency. Menses irregular and scanty. Urine: acid, specific gravity 1008; trace of albumin; no Bence-Jones protein; no sugar; no excess of urobilin; few leucocytes and oxalate crystals in deposit.

Blood.—Hæmoglobin, 93 per cent; red blood-corpuscles, 4,900,000; white blood-corpuscles, 8240; normal differential count. Coagulation and bleeding time, normal. Wassermann reaction, negative.

RADIOGRAMS.—Dr. R. E. Roberts, May, 1932.

Pelvis.—There is no deformity and no increase in size or thickness of the pelvic bones. In the right innominate bone are seen small areas of slightly exaggerated lamellation, together with small translucent areas suggestive of cyst formation.

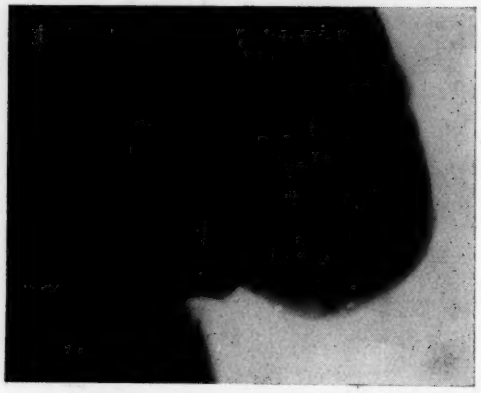
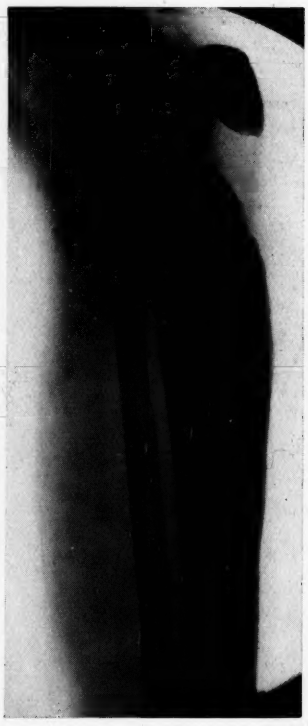


FIG. 271.—Radiogram of mandible.

Lumbar Spine.—The vertebral bodies show no alteration in shape and the intervertebral spaces are normal. The normal texture of the bones has been altered, the upper and lower margins showing a homogeneous slight increase in density, whilst in the central portions are irregular areas of opacity and translucency without any suggestion of lamellation.

Skull (*Figs.* 269, 270).—The calvarium shows a slight thickening with loss of differentiation between the inner and outer tables and diploë. Irregular areas of translucency are seen, some on the outer surface and some extending through the whole thickness of the bone. The base of the skull shows no thickening. The pituitary fossa



FIGS. 272, 273.—Radiograms of left and right tibiæ.

is normal in size, and the sphenoidal and frontal sinuses show normal appearances.

Mandible (*Fig.* 271).—The mandible on both sides shows a marked irregular thickening of both the horizontal and the ascending rami. This is more marked on the right side than on the left. The normal texture is lost and the whole bone presents a coarse opaque basis, throughout which are scattered numerous irregular translucent areas, some of which are clearly defined and suggest cysts. (Very little change has taken place since the previous X-ray examination of Oct. 10, 1930.)

Left Tibia (*Fig.* 272).—The anterior border of the upper two-thirds of the tibia shows a marked subperiosteal thickening which produces a convexity of this border. The medulla is encroached upon slightly by the cortical thickening. There is a slight tendency to the formation of irregular vertical striæ. The tuberosities and lower end of the tibia show normal X-ray appearances, as also does the fibula. Though the anterior tibial border is convex there is no actual bowing of the tibia.

Right Tibia (*Fig.* 273).—In the upper third of the tibia there is a cystic area with fairly well defined upper and lower edges. The cortex in this region is thinned anteriorly, but there is only a very slight expansion; the anterior surface of the bone bulges slightly, but merges with the surface of the bone lower down where the cortex shows slight thickening.

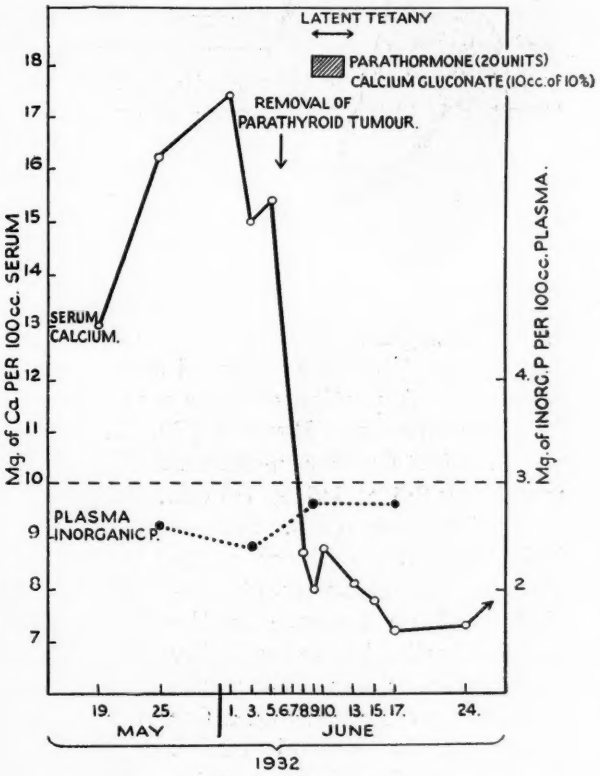


FIG. 274.—Showing calcium content of serum and inorganic phosphorus of plasma before and after removal of parathyroid tumour.

Urinary Tract.—Shadows in left renal area suggestive of numerous small stones and one suggestive of stone in left ureter. Shadow behind symphysis pubis suggestive of small stone in bladder.

CALCIUM AND PHOSPHORUS METABOLISM BEFORE OPERATION (*Fig.* 274).—Hypercalcæmia was constantly present before operation, varying between 13 and 17·4 mgrm. per 100 c.c. The plasma inorganic phosphorus was 2·4 to 2·6 mgrm, non-protein nitrogen was 33 mgrm., and uric acid was 4 mgrm. per 100 c.c. The average calcium excreted per three-day period on a diet containing 150 mgrm. calcium daily was 0·63 grm. in urine and 0·51 grm. in fæces.

Operation (June 6, 1932).—Although there was no apparent swelling in the neck during repose, a distinct fullness was noted in the right side of the neck when it was stretched backwards over the sand-bag before the incision was made. The anæsthetic used was intratracheal ether. After the usual collar incision the infrahyoid muscles were divided transversely, when the fullness previously noted was seen to be due to a swelling immediately below the right lobe of the thyroid gland.

This tumour (the right inferior parathyroid) was oval in shape and measured 2·5 × 1·5 × 1·0 cm. and was slightly lighter in tone than the thyroid gland itself, which it had displaced upwards. It was situated immediately below the right lobe of the thyroid, which covered its upper third. The large mass of tumour lay between the trachea and the carotid sheath. It was completely exposed by turning inwards the right lobe of the thyroid, to which it was attached by a few delicate adhesions; after dissection of these the main blood-vessel supplying the tumour (a small branch of the inferior thyroid) was ligated and the tumour removed.

Fig. 275.—Right inferior parathyroid.

A further search was made for other parathyroid tumours. There was none on the right side, but on turning the left lobe inwards a small tumour was found in the upper part of the left lateral lobe. This was completely buried in the substance of the thyroid and was removed. It proved to be a thyroid adenoma. A portion of bone was removed from the right tibia.

Pathological Report (Dr. H. H. Gleave).—

1. *Right Inferior Parathyroid* (*Fig.* 275).—This is a firm pale fleshy tumour measuring 2·5 × 1·5 × 1·0 cm., weight 2·5 grm.

Microscopic (*Fig.* 276).—This shows an enlarged parathyroid, the enlargement consisting of an increase in the principal cells. These are highly vesiculated. Some of the nuclei are greatly enlarged, and a few cells contain several nuclei. Dark oxyphil cells are scanty. Some of the acini have a central lumen containing acidophilic material.

2. *Small Tumour from Upper Pole of Left Lateral Lobe of Thyroid* (*Fig.* 277).—The tumour measures 1 cm. in diameter. It shows large thyroid vesicles containing thin colloid and lined by flattened cells.

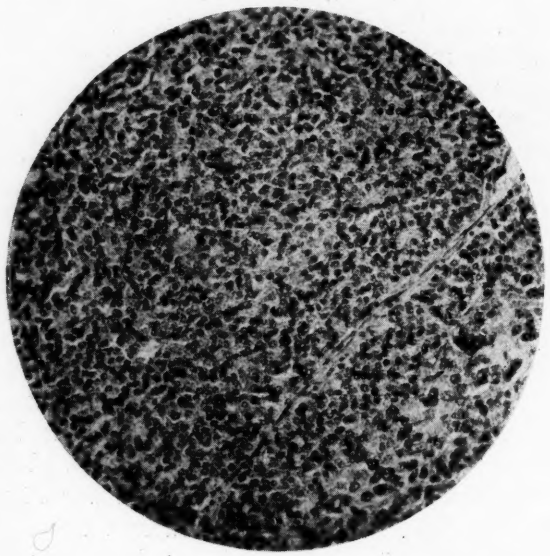


Fig. 276.—Microscopic appearances of parathyroid. (× 140.)

3. *Piece of Right Tibia.*—Specimen is an irregular piece about 1 cm. cube.

Macroscopic.—It has a granular appearance, and cuts easily, but requires decalcification for preparation of sections.

Microscopic.—The structure is almost uniform (*Fig.* 278). Numerous bone trabeculæ, measuring about 50 to 100 μ in width, and in length up to 0·5 mm., are seen lying in a rather greater amount of fibrous tissue. The trabeculæ are frequently surrounded by a narrow zone of osteoid tissue. Osteoblasts, applied to the trabeculæ, are numerous. Osteoclasts are few. They lie both in the fibrous tissue and applied to the trabeculæ. They have very numerous nuclei which fill the cell, and a smaller amount of cytoplasm than usual. The fibrous tissue between the trabeculæ consists of very numerous spindle cells and delicate collagen fibrils. In it are numerous wide capillaries with a wall of a single layer of endothelium, and a few small arterioles.

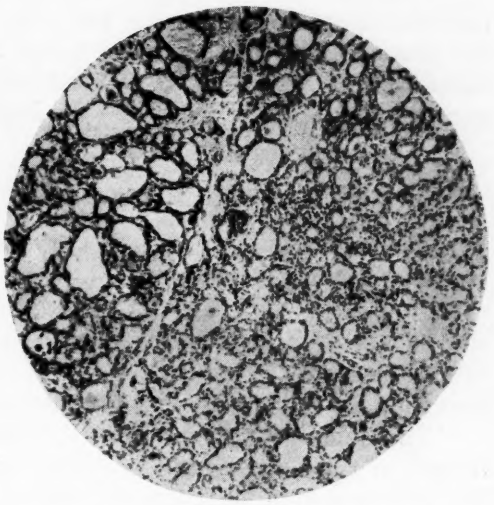


FIG. 277.—Thyroid adenoma. (× 50.)

FIG. 278.—Bone from right tibia. The dark masses are nuclei of osteoclasts.

The condition is thus *osteitis fibrosa.* Formation of imperfect bone is in excess of resorption.

POST-OPERATIVE PROGRESS.—

June 6, 1932.—10 p.m. (eight hours after operation): Comfortable, no evidence of latent tetany.

June 7.—9.30 a.m.: Slight tingling in fingers and toes; no latent tetany demonstrable by muscle irritability. 6 p.m.: No pain or tenderness of affected bones.

June 8.—10 a.m.: Blood calcium 8·7 mgrm. per 100 c.c. No tetany.

June 9.—8 a.m.: Well-marked Chvostek sign, and twitching of left face. No Trousseau's sign. Intravenous calcium gluconate (10 c.c. of 10 per cent solution) and 20 units of parathormone given with relief of tingling and facial twitching. These injections were repeated on the three following days.

June 14.—No recurrence of tetany. Commenced high calcium diet and calcium lactate 20 gr. t.d.s.

Calcium and Phosphorus Metabolism after Operation.—The serum calcium dropped to 8 mgrm. per 100 c.c. after operation (*see Fig.* 274) and the plasma inorganic phosphorus was 2·8 mgrm. A month after operation the average calcium excreted per three-day period on a diet containing 150 mgrm. calcium daily was 0·2 grm. in the urine and 0·45 grm. in fæces.

COMMENT.

The bone changes, the parathyroid tumour, and the pre- and post-operative changes in calcium metabolism are typical. The patient presented, however, one feature which calls for comment. On purely radiological evidence she had previously been regarded as possibly suffering from leontiasis ossea, osteitis deformans, chronic osteomyelitis, etc. This emphasizes again the necessity for full metabolic investigation in cases of generalized bone disease.

The metabolic investigations undertaken in our case proved the true nature of the underlying condition, and, in the light of recorded experience, the presence of a parathyroid tumour was confidently predicted.

ADDENDUM.

Two recent radiographs of the skull and tibia (*Figs.* 279, 280) which are comparable to *Figs.* 270 and 273. Note the increased density of the upper end of the tibia and the increased calcification in the skull with greater clarity of outline, particularly in the vascular markings. (Sept. 19, 1932.)

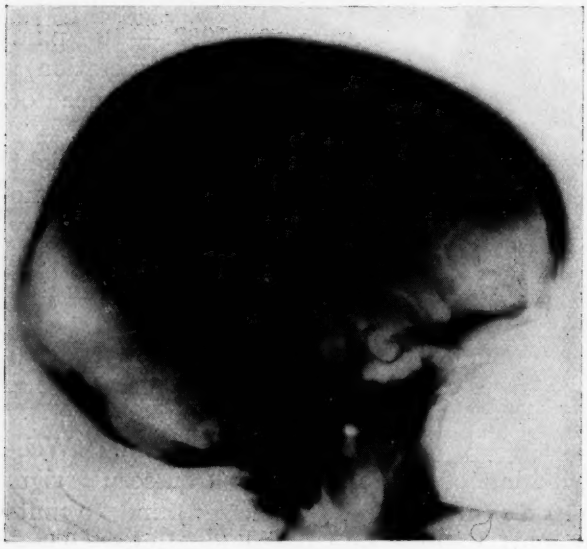


Fig. 279.

Fig. 280.

THE DIAGNOSIS AND TREATMENT OF GENERALIZED OSTEITIS FIBROSA WITH HYPERPARATHYROIDISM.

By R. C. ELMSLIE, F. R. FRASER, T. P. DUNHILL, R. M. VICK, C. F. HARRIS, and J. A. DAUPHINEE,

ST. BARTHOLOMEW'S HOSPITAL, LONDON.

A number of recent publications have shown clearly that there are cases of generalized osteitis fibrosa associated with hyperactivity and tumours of the parathyroid glands, and that in these cases benefit is derived from the extirpation of the parathyroid tumour or tumours. The relevant literature has been reviewed by Barr and Bulger (1930), and in this Journal by Hunter and Turnbull (1931), and will not be referred to in this paper except in particular instances. If the diagnosis of generalized osteitis fibrosa and hyperparathyroidism is made, an operation to explore the parathyroid glands is not only justifiable but is indicated. In recording three further cases of this association of generalized osteitis fibrosa with hyperparathyroidism which have been successfully treated by removal of parathyroid tumours, we wish to stress the diagnostic criteria and the difficulties that may arise in the operative treatment. With this object (1) the histories of the three cases, (2) the bony lesions as determined from clinical and X-ray examinations, which in two of the cases covered periods of six and seven years, (3) the results of the chemical investigations, and (4) the operative treatment and the tumours removed, are described and discussed. Brief reference will be made to a fourth case that was studied in the same manner, and in which no parathyroid lesion was found at operation, because the difference between the findings in this case and in the three in which parathyroid tumours were found, point to some of the essentials for diagnosis.

CASE HISTORIES.

Case 1.*—The patient was a married woman, aged 42 years, engaged in housework and in general work on her husband's fruit farm. Her father died of phthisis; her mother, two sisters, and her husband were in good health. She had measles in infancy, jaundice at the age of 12, and tonsillectomy was performed when she was 22 years of age. She had attacks of severe frontal headaches and nausea all her adult life, at monthly intervals and lasting two days. Dysmenorrhœa had continued since puberty; she had never been pregnant.

In 1920, at the age of 32, pains in the left arm occurred after exertion. In 1921 a fracture of the right humerus above the elbow occurred, and healed quickly. In 1922 there were pains in the shoulders, attributed to neuritis. In 1924 a spontaneous fracture occurred in the right humerus, and she was admitted to the

* The earlier clinical history of this case and reproductions of X-ray photographs of some of the bones were published by one of us (R. C. E.) in the *Robert Jones Birthday Volume* (1928).

Royal National Orthopædic Hospital and kept under observation there after leaving the hospital. Cyst-like expansions were discovered on X-ray examination in several of the long bones. Two of the cysts were curetted. During the following years further cysts appeared. Most of these were curetted as they expanded and seemed likely to lead to fractures. From 1927 onwards, progressive rarefaction of the tibiæ was noted, while the skull became thickened. In 1925 the serum calcium was 9·8 mgrm. per 100 c.c.; in 1927 the serum calcium was 14·6 mgrm. and 14·8 mgrm. per 100 c.c. on two occasions, and the blood phosphorus was 1·6 mgrm. and 1·1 mgrm. per 100 c.c.

She was admitted to St. Bartholomew's Hospital in January, 1930, and was then a small alert woman, looking older than her years, with pains in the limbs, and considerably limited in her activities. The muscles of the limbs were wasted. The skull was not large, but the forehead was prominent and showed definite bossing. The bones of the face were symmetrical, and nothing abnormal was noted in the vertebral column and ribs. The arms and legs showed numerous scars of operations, and the left arm was in plaster because of a recent fracture of the humerus. The long bones had many irregularities in their outlines and there was marked bowing of the humeri and tibiæ. The fingers and toes appeared normal. The alimentary, respiratory, cardiovascular, and nervous systems appeared normal, but the blood-pressure was—systolic 150 mm. of Hg, and diastolic 105. The urine was normal, and X-ray examination of the kidneys failed to show the presence of renal calculi. In the neck the right lobe of the thyroid appeared larger than the left. The results of the operations on the bone cysts and of the X-ray examinations of the bones, and the results of the chemical investigations, are given in later sections.

An operation on the neck was performed on May 8, 1930, and a parathyroid tumour removed from the left side. Symptoms and signs of tetany appeared during the week following the operation, and gradually increased in severity. After four doses of Collip's parathyroid extract, 5 units twice a day, the tetany disappeared and did not recur. Following the operation the patient rapidly grew stronger, and a month later returned home. Six months after the operation she reported that she was suffering from menorrhagia, and still had some pains in the limbs, but no further fractures had occurred. No increase in the density of the bones could be demonstrated, but she was carrying on her normal occupation with greater comfort and efficiency.

She was admitted for a few days in May, 1932, two years after the removal of the parathyroid tumour. She was in good health, free from pain or other symptoms, and working hard on her farm. There was no striking change on physical examination, except that the blood-pressure had risen to systolic 220 mm. of Hg, diastolic 130. There was no increase in body weight. X-ray examination of the bones showed that definite improvement had occurred in them, and these changes are described in a later section. The serum calcium was 11·0 mgrm. per 100 c.c., and the serum phosphorus 3·2 mgrm., which are normal values. She states that she is frequently subjected to traumata that would previously have caused fractures but now have no effect.

Case 2.—The patient was an unmarried woman of 26 years, who worked as a clerk in the Civil Service. Her family were healthy, and she had had no serious illnesses, but had never been robust.

In December, 1928, an attack of influenza was accompanied by aching of the bones, which persisted and grew steadily worse, was increased by exercise and improved with rest. In May, 1929, a tumour, described as a fibrous epulis, was removed from her jaw. In January, 1930, wasting of the muscles of the shoulder and pelvic girdles was noted and considered to be due to a myopathy. In July, 1930, a fracture of the head and neck of the femur followed a slight injury, and she was admitted to St. Bartholomew's Hospital.

She was a thin nervous woman, free from pain when at rest in bed. The alimentary, respiratory, cardiovascular, and nervous systems appeared normal. A bacilluria due to *B. coli* was present, and X-ray examination of the kidneys some months later showed calculi in the pelves of both kidneys. The left humerus was

expanded in the upper third, and the outline of the right tibia was irregular; the fracture of the left femur appeared to be healing satisfactorily. A small localized swelling was seen and felt near the lower pole of the right lobe of the thyroid gland. The results of the X-ray examination of the bones and of the chemical investigations are described in later sections.

An operation on the neck was performed on Sept. 15, 1930, and a parathyroid tumour was removed from the right side. The recovery was uneventful. A month after the operation severe pain developed in the right leg, but no evidence of fracture was obtained, and the pain gradually subsided. Pains in the bones persisted for some months but gradually lessened, and six months after the operation they were only present on considerable exertion. Menstrual loss was increased, but the patient was gaining weight and strength. In June, 1931, she had so improved in general health and in strength that she applied for reinstatement in her post in the Civil Service. X-ray examination showed definite increase in the density of the bones.

In May, 1932, twenty months after the removal of the parathyroid tumour, she felt well and appeared healthy. She had regained her muscular strength and had put on nearly a stone in weight. A few days previously she had slipped in the street and fallen without any fracture resulting. X-ray examination of the bones showed striking improvement. The changes in the bones following the operation are described in a later section.

Case 3.*—The patient was an unmarried girl, aged 23 years. Her parents and nine brothers and sisters were alive and healthy, and she had been free from serious illness in infancy and childhood.

When 15 years old, a pain developed in the left leg that caused her to limp. She was admitted to a hospital, and extension applied. Following this she was admitted to five different hospitals on several occasions, where the treatment adopted in most instances was the putting of the left leg in plaster; the left femur had sustained a spontaneous fracture at the age of 16. At this time the right leg became painful and a spontaneous fracture occurred in the right femur, and when she was 17 years old, swellings appeared in the fingers, wrists, and sternum. When 18 years old, the upper part of the left tibia was much enlarged and a pulsating swelling over its inner aspect was diagnosed as an osteoclastoma. Later the back became bent, and a swelling appeared in the left humerus, but the enlargement of the head of the left tibia had ceased to pulsate. When 22 years old, the left thumb and left femur were curetted at the Royal National Orthopædic Hospital, and a year later a spontaneous fracture occurred in the left humerus. During most of her illness she had been bedridden. She complained of few symptoms apart from the bones; the digestion was good; there were no symptoms of urinary trouble; menstruation had been normal, but became irregular and then ceased while under our observation in hospital.

She was admitted in September, 1930. The skeleton was grossly deformed, and there was much muscular wasting. The skull was irregularly enlarged, the face appearing small below it. The spine was twisted and shortened. The thorax showed a long antero-posterior diameter, the sternum was prominent and its surface irregular, and the lower ribs splayed out. The long bones were bowed, and had irregular expansions at the extremities and along the shafts. The fingers were similarly irregular and thickened. She was bright, intelligent, and co-operative. No disease of the nervous system, heart, or lungs was discovered. The lower pole of the left kidney was palpable; the urine contained albumin and many pus-cells, and X-ray examination showed calculi in the pelves and calices of both kidneys. Urea clearance tests gave a standard clearance of 18·5 c.c. and a maximal clearance of 34·5 c.c. of blood per minute, as compared with the normal figures of 54 c.c. and 75 c.c. The basal metabolic rate was −22 per cent. Blood-count: red blood-cells,

* The earlier clinical history of this case and reproductions of X-ray photographs of some of the bones were published by one of us (R. C. E.) in the *Robert Jones Birthday Volume* (1928).

5,100,000; white blood-cells, 9,500; hæmoglobin, 92 per cent. The results of the operations on the bones and of the X-ray examinations of the bones, and the results of the chemical investigations, are described in later sections.

On Jan. 19, 1931, an operation was performed on the neck and a parathyroid tumour removed. Two days after the operation symptoms of tetany appeared. Injections of Collip's parathyroid extract were given for the following five days in doses of 10 units daily, when the symptoms of tetany disappeared, and calcium lactate, by the mouth, and radiostoleum were given for a further period of some weeks. Sixteen days after the operation the patient was discharged from hospital, but returned five months later for further observation. During this interval she gained considerably in strength and felt better. There was no change in outward appearance; X-ray examination of the bones and kidneys showed no change; pus was still present in the urine; and the urea clearance tests showed no improvement in renal function.

She was seen again in May, 1932, sixteen months after the removal of the parathyroid tumour. She felt well and was leading an active life at home. She seemed happy and had gained nearly a stone in weight. There was little change in the appearance of deformity, but she was able to do without crutches except when walking in the street. Menstruation was normal once more. X-ray examination of the bones showed definite changes that are described in a later section.

EXAMINATION OF THE BONES AND CYSTS.

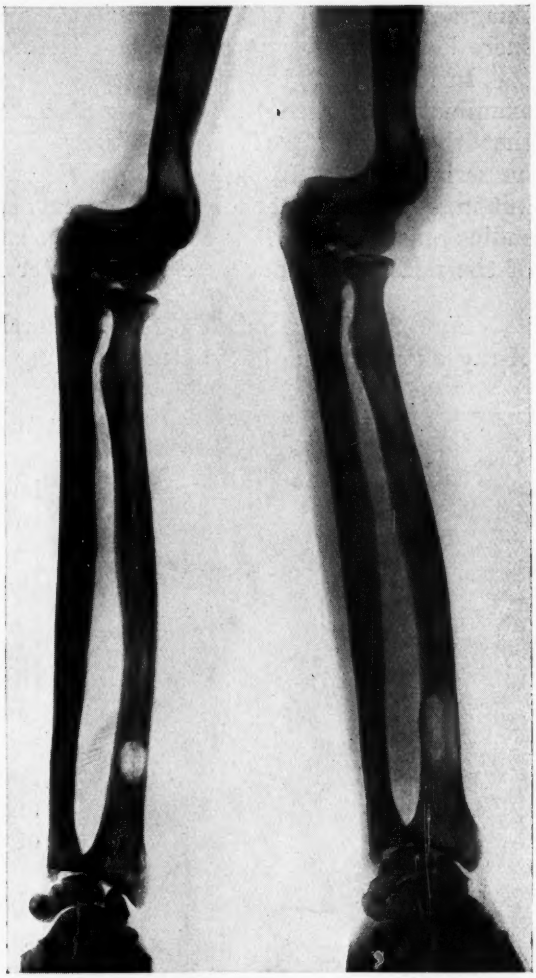
Case 1.—When this patient was admitted to the Royal National Orthopædic Hospital in December, 1924, there was a flail-like condition of the shaft of the right humerus just above the elbow. X-ray examination demonstrated cystic expansions in the right and left humeri, left radius, left femur, and right tibia. In the right humerus the bone was completely absent for a space of about two inches in the lower part of the shaft, and there was a second large cavity showing only a vague bony outline occupying the whole of the upper part of the shaft. The bone between these two cavities was much rarefied.

A cavity in the right tibia was explored for diagnosis, and a simple cyst containing brown fluid found. There were no solid contents and the wall consisted of smooth bone. Three weeks later the two large cysts in the right humerus were opened. In each case the cavity was covered by fibrous periosteum, and it was bounded above and below by the irregular bone of the humerus. The cavities were curetted, so that after the operation the humerus consisted of three portions connected by two periosteal tubes. The arm was supported by a leather case, and the patient returned home. Three months after the operation the bone had consolidated; the arm was out of shape, but had become quite strong and useful. The cysts, however, did not disappear completely; the upper one refractured later and both cysts had enlarged again to a considerable size on X-ray examination in 1930.

From December, 1924, onwards the patient was kept under observation, the cysts in the various bones were watched, and whenever one appeared large enough to suggest the risk of a fracture it was opened and curetted. In every case the cyst thus curetted filled up to some extent and the bone became stronger, but as a rule the cystic condition remained evident on X-ray examination. A large cyst in the upper part of the shaft of the right radius was aspirated without being opened. This did not cause consolidation of

the cyst, which eventually expanded and became very painful. It was finally opened, and after this it filled up and the bone became stronger.

The cysts explored had no solid contents, and with one exception no proper lining. One cyst in the middle of the shaft of the left femur, which had caused severe pain, was lined by a thick grey membrane, which in places showed fibrous tissue and small fragments of bone, but which also showed quite definite areas of organizing blood-clot. The appearances suggested that there had been a recent hæmorrhage into this cyst, and that the lining membrane had been formed by organization of the clot.



A B

FIG. 281.—*Case* 1. Right radius and ulna (A) before, and (B) two years after, removal of the parathyroid tumour, showing increase in density of bone and increase in size of cyst in lower end of radius.

Histological investigation of bone curetted from the margins of the cysts showed in one case that the bone was being absorbed by osteoclasts. Except for this, nothing was found to explain the way in which the cysts were formed, and in no case was there any evidence of decalcification of the bone at the margins of the cysts. No giant-cell tumours were found, nor was there any evidence of fibrosis of the marrow, which appeared normal in the neighbourhood of the cysts. There was nothing in the X-ray photographs to suggest the massive formation of fibrous tissue in the interior of the bones such as is seen in other forms of cystic disease of bone.

During the seven years 1924–30 further cystic changes were found in the right and left humeri, the left radius, the right and left femora, the pelvis, and the right tibia. In the skull the calvarium was found to be much thickened, the maximum being about $\frac{5}{8}$ in. as estimated by an X-ray photograph, and there appeared to be a cavity behind the frontal sinus which was possibly a small cyst. The texture of the bone of the calvarium was obviously abnormal, the X-ray shadow showing a mottled woolly appearance.

X-ray examinations of the shafts of the long bones away from the cysts showed from the first a certain amount of porosis with widening of the

Haversian spaces, and this appearance became much more marked by 1930. There was not thickening of any bone except the calvarium and no bending of the long bones except at the site of the cysts where fractures or crumpling occurred.

Re-examination of the bones by means of X rays, six months after the removal of the parathyroid tumour, showed that the cysts had not altered and that the cystic areas in the right humerus were still very large. Control skiagrams showed that there was still much porosis of the bones, and that there had been no improvement in the bones as seen by X-ray photographs.

In May, 1932, two years after removal of the parathyroid tumour, X-ray examination showed that the bones had improved generally in density and that the cortical parts were thicker. The old cysts in the right and left humeri had filled up to a considerable extent and become much more dense, but many of the cysts were still present, and one in the lower end of the right radius was actually larger than it was before operation, although the shafts of the radius and ulna were much better formed (*Fig.* 281).

Case 2.—On admission to St. Bartholomew's Hospital in July, 1930, X-ray examination showed the following abnormalities of the bones :—

1. Pelvis—extensive areas with formation of cystic spaces.

2. Left femur—head rarefied, a large cyst in the neck through which the bone was fractured, and cystic patches involving the cortical part of the bone in the upper part of the shaft.

3. Right femur—cystic patches in the cortical part of the bone extending through the shaft so that there was practically no normal cortex.

4. Right humerus—cortex rarefied, Haversian canals showing plainly, and cystic cavities involving portions of the cortex.

5. Skull—calvarium thickened to about half an inch and showing mottled appearance.

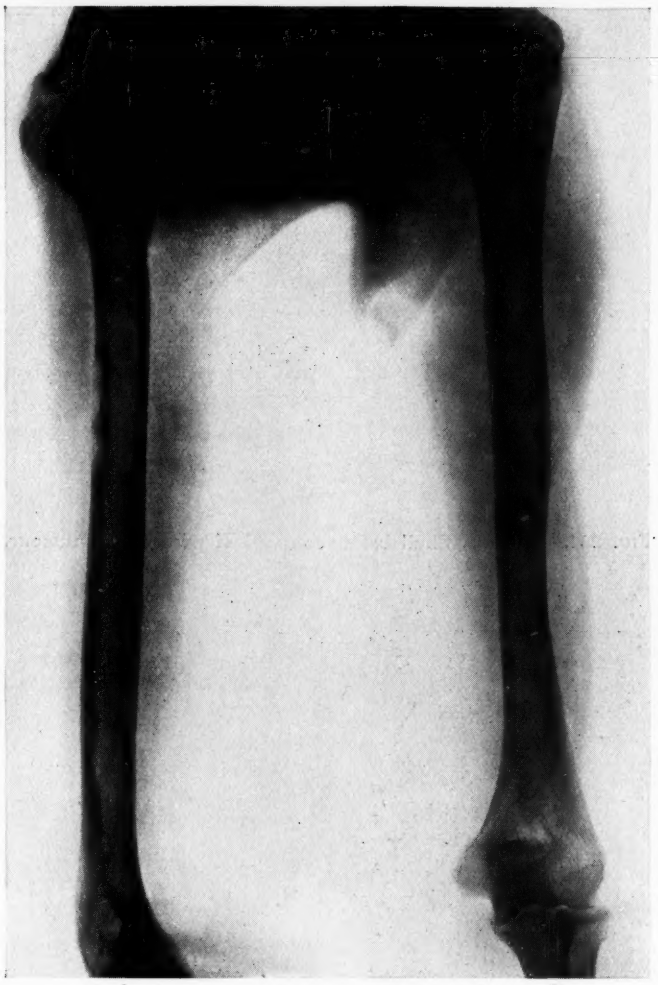
FIG. 282.—*Case* 2. Skiagram showing dense opaque area at site of cyst in left humerus twenty months after removal of parathyroid tumour.

Following the removal of the parathyroid tumour in September, 1930, further X-ray examinations showed a large cavity in the right femur a little below the small trochanter, and, when compared with controls, the bones of the right forearm showed much rarefaction with loss of definite cortex and extensive cystic patches, one of which in the radius caused expansion of the bone.

The patient was re-admitted in June, 1931, nine months after the removal of the parathyroid tumour, when she had improved considerably in health

and strength, and X-ray examinations showed the following changes, and indicated great improvement in the bony condition :—

1. Right femur—the large cystic area in the upper part of the shaft still clearly visible, but covered by a definite cortical layer, and the lower part of the shaft showing much thickening of the surface of the bone.



A B

FIG. 283.—*Case* 2. Right humerus (A, before, and (B) twenty months after, removal of parathyroid tumour, showing disappearance of cystic cavities and increase in cortex and in density of bone generally.

2. Left femur—cystic areas no longer seen, cortex re-formed, but not dense, mottled in appearance, with irregular projections on the surface, and general increase in density of bone.

3. Right humerus—cystic areas no longer seen.

4. Left humerus—oval opacity below surgical neck, and oval expansion without opacity in lower part of shaft.

FIG. 284.—*Case* 2. Skull before removal of parathyroid tumour.

FIG. 285.—*Case* 2. Skull twenty months after removal of parathyroid tumour, showing diminution of mottling, thinning of bone, and greater distinctness of vascular channels.

5. Right tibia and fibula—extensive thickening and opacity of shaft in middle third, the fibula more than twice normal thickness.

In May, 1932, twenty months after the removal of the parathyroid tumour, X-ray examination showed that the cysts were all transformed into dense opaque areas, and the bones well formed and of good density (*Figs.* 282, 283). The right femur, left radius, and right tibia and fibula showed much new periosteal bone. The skull was less mottled and thinner; the outline of all the vascular channels had become distinct (*Figs.* 284, 285).

Case 3.—When this patient developed a limp at the age of 15, X-ray examination showed slipping of the upper epiphysis of the left femur. At the age of 16, when the first spontaneous fractures occurred, skiagrams made at St. Thomas's Hospital showed the following abnormalities :—

1. Tibiæ—very thin in texture and translucent, the right straight, the left a little collapsed at the upper end.

2. Radii—texture fairly good but Haversian spaces more evident than normally, lower ends much enlarged in region of epiphysial line and obviously porous in texture.

3. Ulnæ—texture fairly good but Haversian spaces more evident than normally, epiphysial cartilages widened but without the cupping of the diaphysis which occurs in rickets.

4. Hands—spiky periosteal projections from the surface of the phalanges, small cystic area in base of first phalanx of right thumb.

5. Spine—slight scoliosis, small renal calculi noted.

At the age of 18, X-ray examination at the Pyrford Hospital showed the following further changes :—

1. Left tibia—markedly collapsed in upper part.

2. Femora—very light in texture without any proper cortex, shafts collapsed and deformed.

3. Pelvis—triradiate.

4. Right humerus—upper end collapsed and deformed.

5. Radii and ulnæ—porosis, particularly at lower ends, where epiphysial cartilages were widened.

6. Hands—cyst in first phalanx of right thumb larger, cyst in metacarpal bone of left middle finger, periosteal new bone less marked.

7. Skull—calvarium greatly increased in thickness, appearing mottled and woolly.

At the age of 20 years, the head of the left tibia and the base of the first phalanx of the left thumb were explored and were each found to contain solid tissue with the structure of an osteoclastoma and a spindle-celled stroma containing a few islets of bone. The bone at the margin showed definite lacunar absorption by giant cells.

On admission to St. Bartholomew's Hospital at the age of 23 years in September, 1930, the condition of the bones was much the same as that already described, allowing for a little increase in the deformities.

On re-admission, three months after the removal of the parathyroid tumour, at a time when she had gained in strength and felt better, there was no change in the appearance of the bones on X-ray examination.

FIG. 286.—*Case* 3. Right femur and part of pelvis (A) before, and (B) sixteen months after, removal of parathyroid tumour, showing increased density of bone surrounding the cystic cavities.

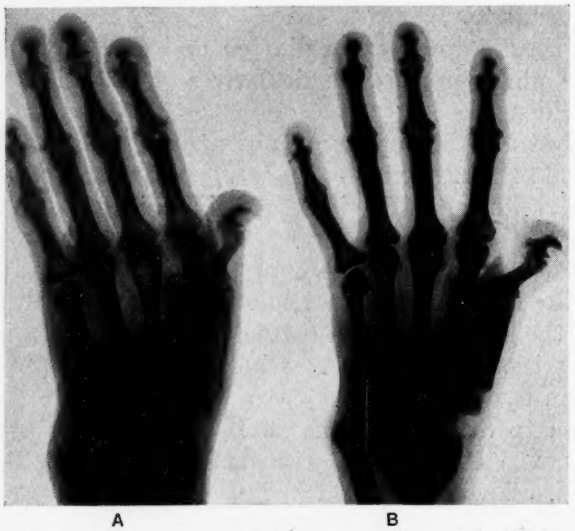


FIG. 287.—*Case* 3. Left hand (A) before, and (B) sixteen months after, removal of parathyroid tumour, showing increase in density of bones generally and opaque area at site of cyst in third metacarpal bone.

In May, 1932, sixteen months after the removal of the parathyroid tumour, X-ray examination showed that the large cystic cavities in both femora still existed but were surrounded by denser bone (*Fig.* 286). The upper ends of the tibiæ had thickened and consolidated and consisted of rather irregularly dispersed cancellous bone. The cortex of the shaft of these bones was also thicker. A cyst in the third left metacarpal bone had been transformed into an opaque area (*Fig.* 287).

CHEMICAL INVESTIGATIONS.

Methods.—During the periods of study the diets of the patients were kept constant, and the calcium and phosphorus contents of the diets were calculated from the tables of Sherman (1924), confirmatory analyses being made in many instances. In most periods the quantities were those present in the patient's ordinary diet, about 1 grm. of calcium and 1·25 grm. of phosphorus a day, so that the relation of intake to output under conditions similar to those of their home life could be estimated. During certain of the periods diets low in calcium, containing 0·25 grm. or less of calcium, were given in order to make a comparison with the observations of other workers. The diets selected were given over a period of four or five days before any collection of excreta was made, in order that conditions should have time to become stabilized. The fæces were collected in four-day periods marked off by carmine. The urine was collected in two-day periods. The acid-base ratio of the diets was kept constant.

The calcium in the serum was estimated by Clark and Collip's (1925) modification of the method of Kramer and Tisdall (1921, 1923), the inorganic phosphorus in the serum by the method of Fiske and Subarrow (1925). The stools were ashed, following the method of Stolte (1911), the ash dissolved in dilute hydrochloric acid with the aid of heat, and the solution used for the determination of calcium and phosphorus. The urine was evaporated and ashed for the estimation of calcium; the inorganic phosphorus was estimated directly in the forty-eight-hour specimens. In *Case* 3 the organic phosphorus in the urine was also measured. This proved to be so small an addition, that the omission of this measurement in the other cases cannot materially affect the results.

Results.—The results of the estimations are given in *Tables I, II, III*, and shown graphically in *Figs.* 288–290. In *Fig.* 291, the calcium and phosphorus balances of each of the three patients are averaged for the periods before and for the periods after the removal of the parathyroid tumour. In the same figure are included a normal balance recorded by Sherman (1924), and the balance in a case of generalized osteitis fibrosa with hyperparathyroidism recorded by Bulger, Dixon, and Barr (1930).

The results of the estimations before operation can be stated briefly as follows:—

The serum calcium (*Table I*) was abnormally high in two (*Cases* 1 and 2) out of the three patients. In *Case* 3 the serum calcium did not rise much above what may be considered a high normal value during the time the patient was under controlled observation, but an abnormally high value had

Table I.—Results of Calcium and Phosphorus Estimations.

Case I.					Case II.					Case III.				
Date	Diet		Serum		Date	Diet		Serum		Date	Diet		Serum	
	Calcium	Phosphorus	Calcium	Phosphorus		Calcium	Phosphorus	Calcium	Phosphorus		Calcium	Phosphorus	Calcium	Phosphorus
	Grm. daily		Mgrm. per cent			Grm. daily		Mgrm. per cent			Grm. daily		Mgrm. per cent	
6/2/30	Normal adult		14·9	2·0	23/7/30	Normal adult		16·4*	—	13/10/30	Normal adult		15·2*	—
13/2/30	1·040	1·259	14·7	2·2	7/8/30	Normal adult		17·7*	—	27/10/30	0·753	1·092	12·7	2·1
20/2/30	1·040	1·259	16·2	2·6	14/8/30	1·022	1·262	15·6	2·5	14/11/30	0·099	0·572	12·1	1·35
10/3/30	0·250	0·800	15·8	2·0	21/8/30	1·022	1·262	15·5	2·2	17/1/31	Normal adult		11·8	2·6
17/3/30	0·250	0·800	15·6	2·8	15/9/30	Operation		—	—	19/1/31	Operation		—	—
8/5/30	Operation		—	—	16/9/30	Uncontrolled		12·4*	—	21/1/31	Normal adult		7·3	2·3
26/5/30	0·986	1·053	5·9	2·9	18/9/30	Uncontrolled		11·2	1·9	21/4/31	0·753	1·092	9·1	4·3
2/6/30	0·986	1·053	5·4	4·0	23/9/30	Normal adult		7·5	2·3	11/5/31	0·099	0·528	9·3	4·1
15/12/30	Normal adult		8·2	—	29/9/30	Normal adult		7·0	3.0	—	—	—	—	—
—	—	—	—	—	16/10/30	1·030	1·297	8·8	2·7	—	—	—	—	—
—	—	—	—	—	30/6/31	Normal adult		10·4	3·6	—	—	—	—	—

* Observations made by others than the present writers.

been noted previously. The amount of calcium taken with the food had no effect on the level of serum calcium in these cases.

The serum phosphorus was abnormally low in all three cases.

On a high calcium diet (*Table II* and *Fig.* 291) the patients stored calcium in *Cases* 1 and 3, but in *Case* 2 there was a negative balance, the

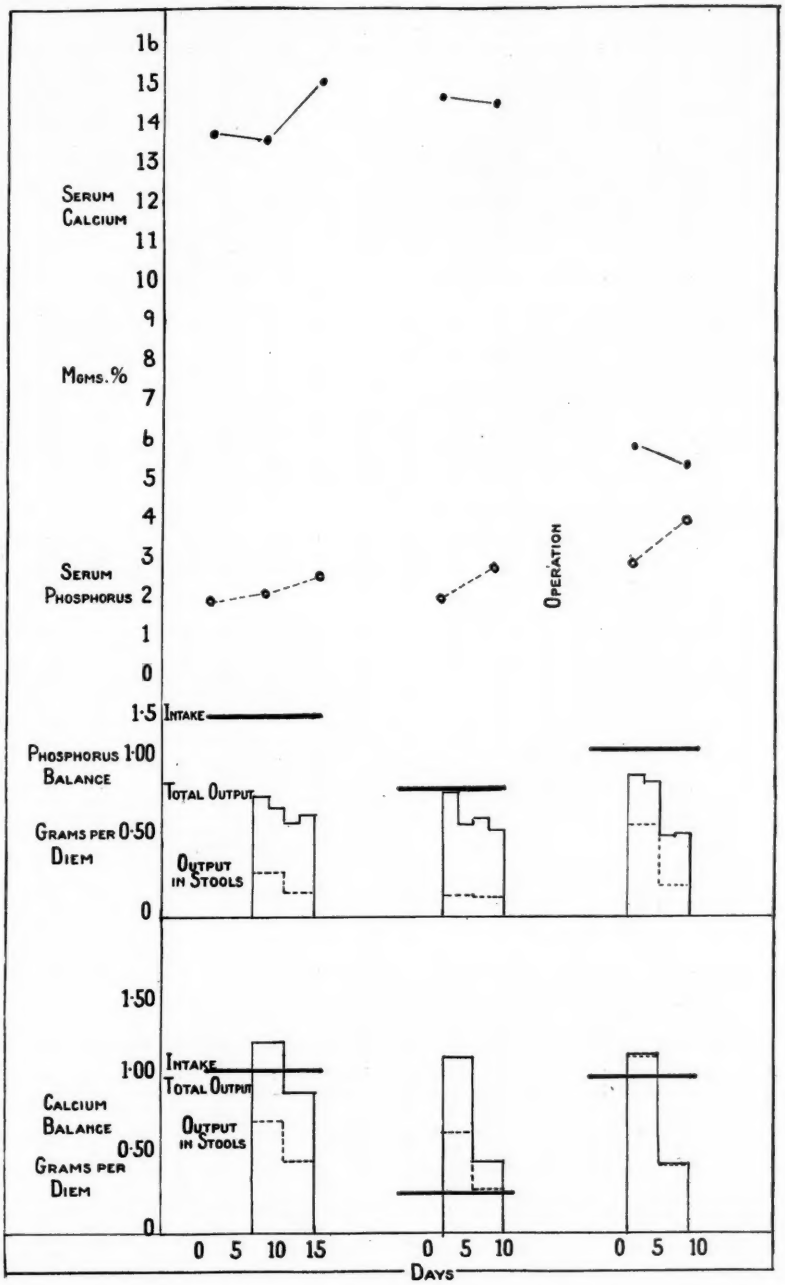


Fig. 288.—*Case* 1. Graphic representation of the calcium and phosphorus metabolism over two periods of eight days before parathyroidectomy and one eight-day period after.

Table II.—HIGH CALCIUM INTAKE.

CASE	4-DAY PERIOD	SERUM		CALCIUM					PHOSPHORUS				
				Intake Daily	Output Daily			Balance	Intake Daily	Output Daily			Balance
		Calcium	Phosphorus		Fæces	Urine	Total			Fæces	Urine	Total	
		Mgrm. Per cent	Mgrm. Per cent	Grm.	Grm.	Grm.	Grm.		Grm.	Grm.	Grm.	Grm.	
		14·9	2·0	—	—	—	—	—	—	—	—	—	—
	1	14·7	2·2	1·040	0·709	0·454	1·163	−0·123	1·259	0·272	0·476	0·748	+0·511
	2	16·2	2·6	1·040	0·462	0·430	0·892	+0·148	1·259	0·150	0·465	0·615	+0·644
I		Operation											
	3	5·9	2·9	0·986	1·121	0·017	1·138	−0·152	1·053	0·570	0·286	0·856	+0·197
	4	5·4	4·0	0·986	0·425	0·010	0·435	+0·551	1·053	0·190	0·315	0·505	+0·548
	1	15·6	2·5	1·022	0·446	0·483	0·929	+0·093	1·262	0·150	0·972	1·122	+0·140
	2	15·5	2·2	1·022	1·012	0·465	1·477	−0·455	1·262	0·404	1·039	1·443	−0·181
II		Operation											
		7·0	3·0	—	—	—	—	—	—	—	—	—	—
	3	—	—	1·030	0·319	0·036	0·355	+0·675	1·297	0·154	0·346	0·500	+0·797
	4	8·8	2·7	1·030	0·542	0·028	0·570	+0·460	1·297	0·320	0·478	0·798	+0·499
	1	12·7	2·1	0·753	0·752	0·159	0·906	−0·153	1·092	0·350	0·322	0·672	+0·420
	2	12·1	2·3	0·753	0·374	0·164	0·538	+0·215	1·092	0·157	0·457	0·614	+0·478
	3	—	—	0·753	0·374	0·160	0·534	+0·219	1·092	0·157	0·480	0·637	+0·455
	4	—	—	0·753	0·326	0·221	0·547	+0·206	1·092	0·143	0·656	0·799	+0·293
		12·1	1·4	—	—	—	—	—	—	—	—	—	—
III		Operation											
		7·3	2·3	—	—	—	—	—	—	—	—	—	—
	5	9·1	4·3	0·753	0·105	0·034	0·139	+0·614	1·092	0·075	0·545	0·620	+0·472

III		12·1 Operation 7·3	1·4 2·3	—	—	—	—	—	—	—	—	—	—
	5	9·1	4·3	0·753	0·105	0·034	0·139	+0·614	1·092	0·075	0·545	0·620	+0·472
	6	—	—	0·753	0·155	0·031	0·186	+0·567	1·092	0·098	0·605	0·703	+0·399
		9·3	4·1	—	—	—	—	—	—	—	—	—	—

Table III.—Low Calcium Intake.

Case	4-day Period	Serum		Calcium					Phosphorus				
				Intake Daily	Output Daily			Balance	Intake Daily	Output Daily			Balance
		Calcium	Phosphorus		Fæces	Urine	Total			Fæces	Urine	Total	
		Mgrm. Per cent	Mgrm. Per cent	Grm.	Grm.	Grm.	Grm.		Grm.	Grm.	Grm.	Grm.	
I	a	15·8	2·0	0·250	0·683	0·431	1·114	−0·864	0·800	0·143	0·515	0·658	+0·142
	b	15·6	2·8	0·250	0·262	0·187	0·449	−0·199	0·800	0·133	0·438	0·571	+0·229
III	a	12·1	1·4	0·099	0·099	0·148	0·247	−0·148	0·528	0·118	0·454	0·572	−0·044
	b	—	—	0·099	0·114	0·143	0·257	−0·158	0·528	0·114	0·362	0·476	+0·052
		Operation											
		9·1	4·3	—	—	—	—	—	—	—	—	—	—
	c	—	—	0·099	0·036	0·017	0·053	+0·046	0·528	0·055	0·344	0·399	+0·129
	d	9·3	4·1	0·099	0·051	0·015	0·066	+0·033	0·528	0·115	0·360	0·475	+0·053

patient excreting more calcium than she was receiving. On low calcium diets (*Table III*) negative balances were present in *Cases* 1 and 3. These diets cannot be considered adequate in calcium intake, and provided a more severe test of the ability to conserve calcium. When, however, the calcium excretions in the urine and in the fæces are considered separately, it is seen that in *Cases* 1 and 2 the urinary excretions are abnormally high on both high and low intakes, since the normal urinary excretions of patients in bed on average diets seldom exceed 0·1 grm., and that in *Case* 3, although the urinary output on the higher calcium diets might be regarded as within normal limits (*Table II*), the urinary output remained high when the intake was greatly reduced (*Table III*). The study of the calcium balance demonstrated the presence of abnormal calcium metabolism, therefore, in *Case* 2 by a negative calcium balance and abnormally high urinary calcium excretion, in *Case* 1 by the abnormally high urinary calcium excretion, and in *Case* 3 by the abnormally high urinary calcium excretion when on a low calcium intake.

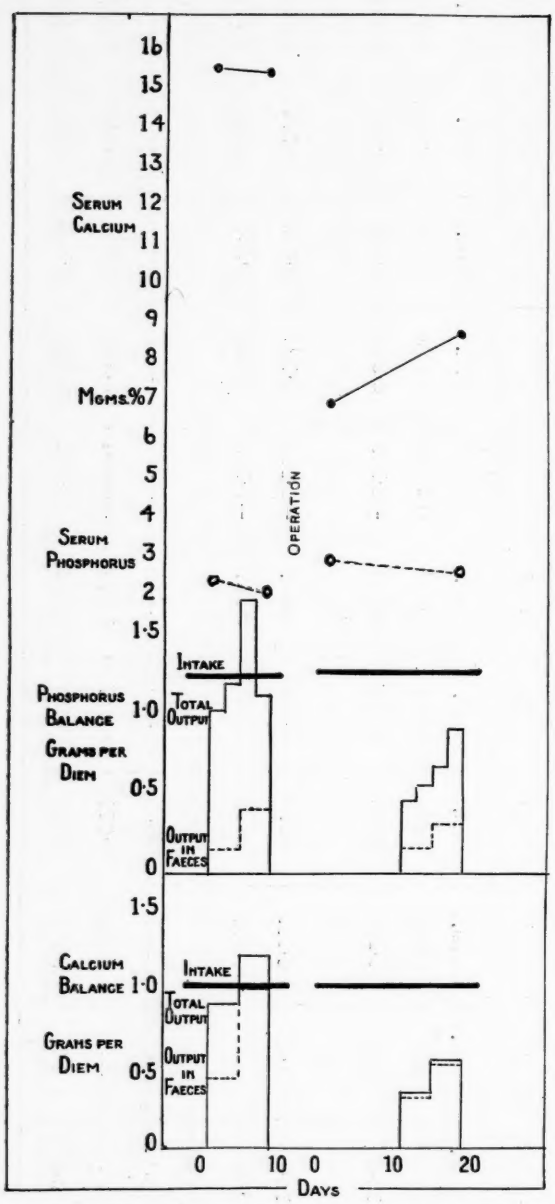


FIG. 289.—*Case* 2. Calcium and phosphorus metabolism as represented in *Fig.* 288.

The study of the phosphorus balance (*Tables II* and *III*) showed a negative balance in *Case* 2 only, phosphorus retention occurring in *Cases* 1 and 3, even when a diet low in both calcium and phosphorus produced a negative calcium balance.

The results of the estimations after operation can be stated briefly as follows :—

The serum calcium (*Table I*) fell in all three cases following the removal of the parathyroid tumour, and the new level was considerably below the normal. In *Case* 3, where the level was not abnormally high before operation, the level after operation was about the same as in the other cases

where an abnormally high value had been found before operation. The rate of the fall varied, for in *Case* 2 the level was still high on the first and third days after parathyroidectomy, and almost reached its lowest level in eight

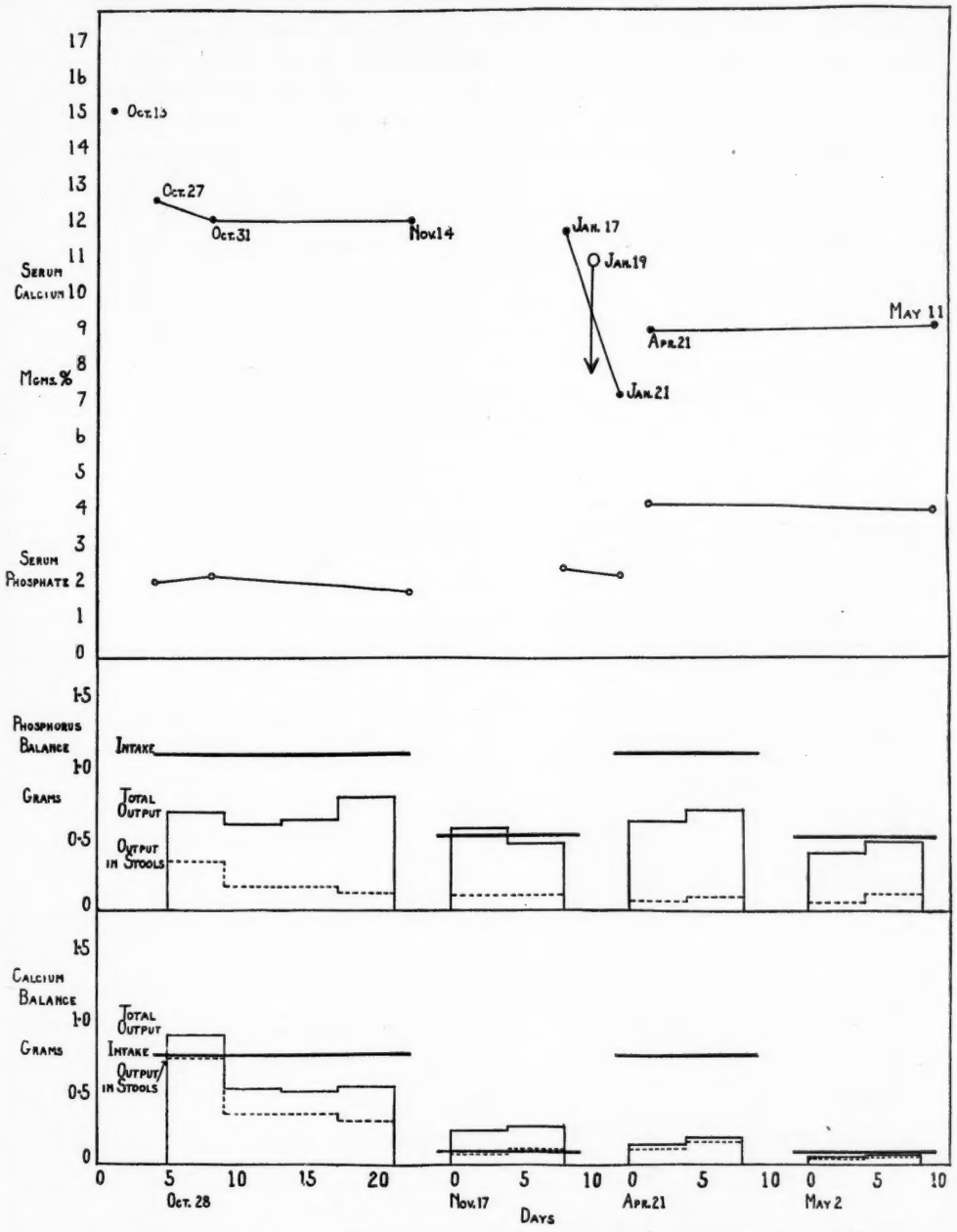


FIG. 290.—*Case* 3. Calcium and phosphorus metabolism as represented in *Fig.* 288.

days, while in *Case* 3 the lowest level was reached within two days after the operation. In *Case* 1 no observations were made in the days immediately following the operation.

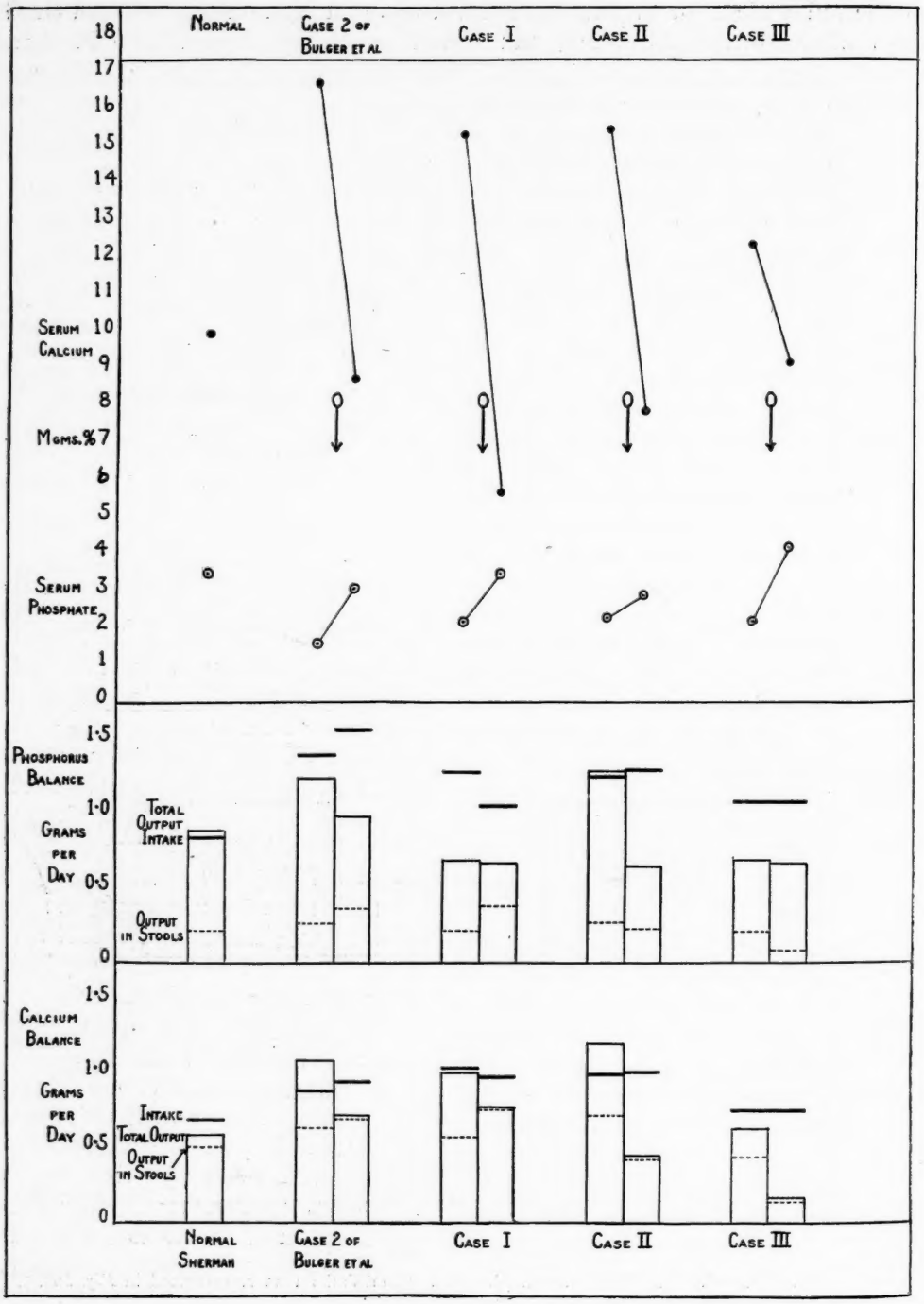


FIG. 291.—Showing the average intake and output of calcium and phosphorus before and after operation in these three cases. For comparison, a similar case by Bulger et al., and a normal calcium and phosphorus balance (Sherman), are included.

The phosphorus in the serum (*Table I*) rose after the removal of the the tumour; the rate of the increase was slow, and only in *Case* 3 had it reached a normal level within three months.

The study of the calcium metabolism (*Tables II* and *III*) showed that there was an increased retention of calcium as a result of the operation, even when there had been no demonstrable loss of calcium before the operation; the increase was greatest when a pre-operative negative balance became positive. In all three cases the excessive excretion in the urine disappeared after the operation, and the calcium excretion was mainly carried out by the intestine.

The phosphorus metabolism (*Tables II* and *III*) was little altered by the removal of the tumours in any of the cases. In *Case* 2 a slightly negative balance became positive, but the absence of any marked change in the phosphorus metabolism is noteworthy in view of the definite changes in calcium metabolism. It will be noted further that two of the patients were able to store phosphorus even while they were losing calcium. The balance data of one of the cases recorded by Bulger, Dixon, and Barr (1930), in which similar findings were made, are included in *Fig.* 291.

OPERATIONS ON THE PARATHYROID GLANDS.

In patients with generalized fibrocystic disease, even when the evidence is sufficiently clear to justify the assumption that a parathyroid tumour is present, this is not always easily found at operation. In none of the three cases under review did the tumour occupy a position usually occupied by a parathyroid gland (*Fig.* 292).

On examination of the region of the thyroid gland in *Case* 1, the lower pole of the left lobe was slightly more prominent than that of the right side. The operation was performed on May 8, 1930. After displaying the thyroid gland, the left lobe was dissected cleanly on its anterior and lateral surfaces. The vessels attached to its postero-lateral margin were ligated and divided to enable the lobe to be lifted and rotated towards the midline, and thus permit examination of the posterior surface. No tumour was found in the neighbourhood of the sites usually occupied by the superior

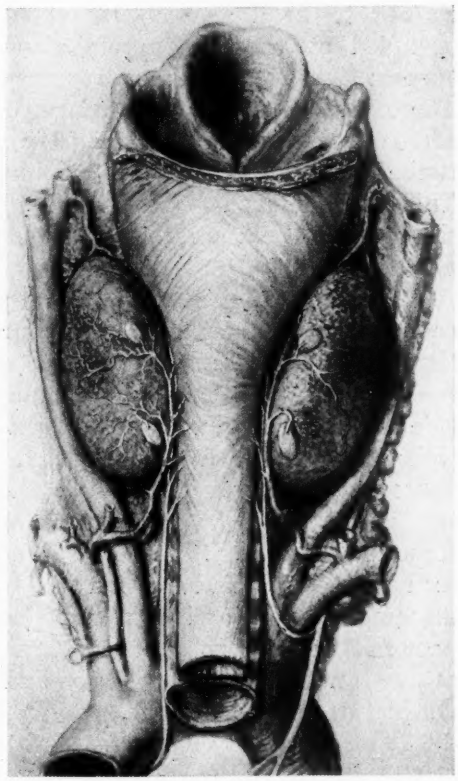


FIG. 292.—Showing the parathyroid glands on the posterior aspect of the thyroid gland (from an article by W. F. Reinhoff in Lewis's *Practice of Surgery* (1929), vol. vi).

or inferior parathyroid glands. The lower pole of the thyroid was slightly but definitely enlarged, and its consistency was such that it obviously contained a nodule. The surface in this region was smoothly continuous with that of the thyroid gland. It was considered that the enlargement might be due to an adenoma, an involution nodule, a cyst, or—conceivably—a parathyroid tumour. So little did it differ from the appearance commonly seen in a thyroid gland that it was left for the moment and the search for a parathyroid tumour continued on the opposite (right) side. This lobe was turned forward in a similar manner, and the regions of the superior and inferior parathyroid glands were examined. No tumour was found, nor was there anything abnormal in the shape or consistency of this lobe. In the absence of a parathyroid tumour elsewhere, and the almost certain knowledge that one must be present, unless the interpretation of the bone changes and the investigation of the calcium metabolism were wrong, the lower pole of the left lobe, with the nodule contained within it, was excised (*see Fig.* 293). Subsequent histological examination showed that the nodule was a parathyroid tumour (*see Figs.* 294, 295). An attack of tetany on the following day gave further confirmation of its nature.

In the second patient, *Case* 2, a slight boss was palpable before the operation on the anterior surface of the mid portion of the right lobe. This proved to be a simple cyst about 1 cm. in diameter. The sites usually occupied by the parathyroid glands were examined without revealing any tumour. A slight fullness was then seen medial to the right carotid artery. The level was about the junction of the middle and upper third of the lobe of the thyroid. On retracting the carotid sheath, and dissecting the areolar tissue over and around the prominence, a tumour gradually became defined. This was removed (*see Fig.* 296). Histological examination of this tumour showed it to be a parathyroid tumour (*see Fig.* 297). No tetany followed this operation.

The operation in *Case* 3 was performed on January 19, 1931. In this case also the parathyroid tumour might easily have been missed. The thyroid gland was of normal size and appearance. The posterior aspect of each lobe was accessible and readily examined. The carotid artery on each side lay normally and no protrusion showed such as was seen in *Case* 2. The right carotid sheath was retracted and the region behind it examined, and subsequently the left. Behind the left carotid sheath, high up, lying on the prevertebral muscles, the rounded end of a smooth, reddish-brown structure was seen. The upper end of this could not be seen even with firm retraction, and it had to be delivered by feel. The tumour was plummet-shaped and flattened (*see Fig.* 298). Microscopic examination showed this to consist of parathyroid tissue (*see Fig.* 299). An attack of tetany on the following day—and continuing for several days—confirmed the nature of the tumour.

Variability in Position. — Halsted and Evans (1907), Lorin and Cuneo (1921), and others have shown that the positions occupied by the parathyroid glands may vary within a wide range, but it was scarcely to be expected that in three successive cases the situation of a tumour arising in one of these glands should be so unusual. This may be the reason for the occasional

failure to find a parathyroid tumour when the evidence of its presence seems conclusive, as in the cases of Barrenscheen and Gold (1928), and of Richardson, Aub, and Bauer (1929).

A further point of interest in connection with the first patient is that, as the tumour was embedded in the thyroid, it must happen occasionally that a normal parathyroid will be sacrificed in the operation of partial thyroidectomy. In this patient the parathyroid, if of normal size, would not have been saved by stripping back the capsule, or shaving off the posterior border of the gland.

DESCRIPTION OF THE PARATHYROID TUMOURS.

Case 1.—

Macroscopic.—The tumour (*Fig.* 293) measures 3 × 1·5 × 1·5 cm. It is irregularly oval in shape, and its colour is yellowish red. It is encapsulated, and thyroid tissue is adherent to the capsule in several places. Fibrous trabeculæ arising from the capsule traverse the tumour, and many cystic spaces are present.

Microscopic.—The tumour (*Fig.* 294) contains obvious spaces of irregular shape, these accounting for approximately one-fourth of its total bulk. It is surrounded by a capsule which along one side and one end divides to include thyroid gland tissue. From the capsule fibrous trabeculæ enter and divide up the gland (*Fig.* 295). The gland tissue is well supplied with blood-vessels—large, small, and capillary. There are also blood-spaces

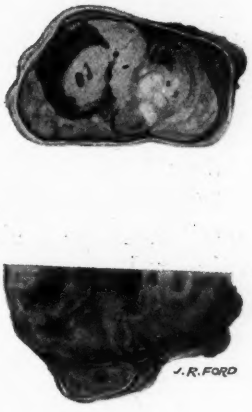
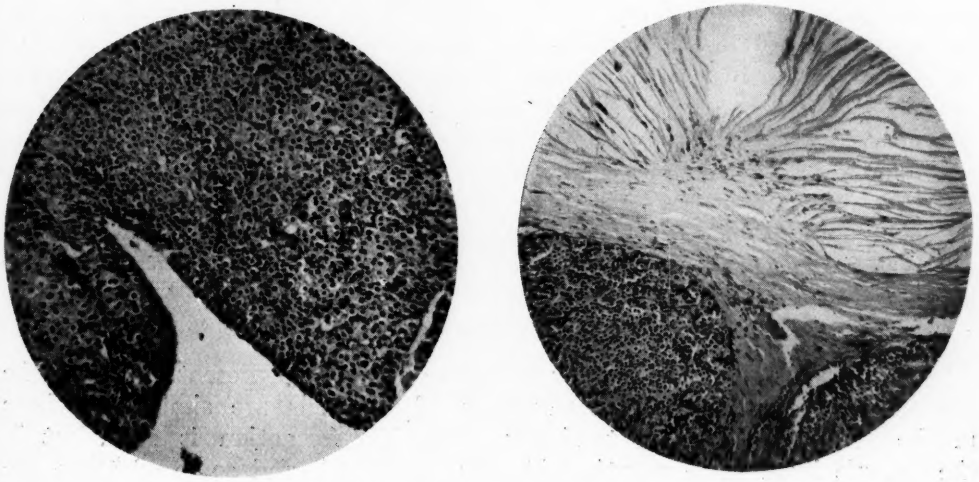


FIG. 293.—*Case* 1. External appearance and cut surface of the tumour. (*Actual size.*)



FIGS. 294, 295.—*Case* 1. Microphotographs of sections of the parathyroid tumour (hæmatoxylin and eosin).

in which blood is only separated from the parenchyma of the gland by flattened cells.

The tumour cells form a fairly uniform field in that there is no suggestion of columns and no attempt at vesicle formation. The individual cells are fairly clearly defined, and vary greatly in shape and size, and many of the cells contain a double nucleus. The cytoplasm is slightly but definitely basophilic, and nowhere can the oxyphile cells of Welch—so commonly found in the normal parathyroid—be found. Many cells are vacuolated, this and the irregularity in size of the nuclei being a conspicuous feature of every field. As the vacuolation advances, the cell becomes relatively large and very clear, with the nucleus still remaining densely stained. The tumour therefore consists of the 'chief' cells of Welch in which multiplication and vacuolation are taking place throughout the tumour.

The cystic spaces appear not to have a lining membrane. Some of them contain fibrin; towards the edges this fibrin is becoming organized, and it is continuous with the fibrous trabeculæ of the tumour. There is very little intercellular stroma, the cells being closely packed.

Case 2.—

Macroscopic.—The tumour (*Fig.* 296) measures $2\frac{1}{2} \times 2 \times 2$ cm. It also is oval in shape and encapsulated. It is yellowish in colour, the vessels showing red on the surface. On section the tumour is solid.

FIG. 296.—*Case* 2. External appearance and cut surface of the tumour. (*Actual size.*)

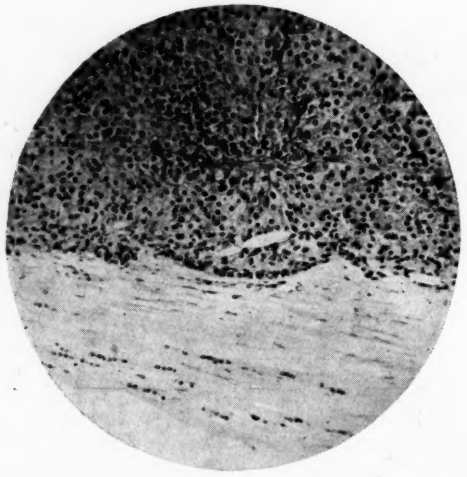


FIG. 297.—*Case* 2. Microphotograph of section of the parathyroid tumour (hæmatoxylin and eosin).

Microscopic.—In this tumour no cystic spaces are present. Within the capsule the character and arrangement of the cells are similar to those in the first case, and no tendency to vesicle formation is seen, but in the capsule itself some parathyroid cells are found, and some of these are arranged in single or double columns and vesicles (*Fig.* 297). No oxyphile cells are seen.

Case 3.—

Macroscopic.—The tumour (*Fig.* 298) measures 3·5 × 1·5 × 0·7 cm. Its shape is that of a flattened plummet. Its colour is dark yellowish brown. It contains much subcapsular hæmorrhage. This was probably caused by the difficulty in delivering the tumour owing to its depth combined with the high situation of its upper pole.

Microscopic.—This tumour differs from the other two in that much more stroma is present, and the cells over a wide extent tend to be grouped

FIG. 298.—*Case* 3. External appearance and cut surface of the tumour. (*Actual size.*)

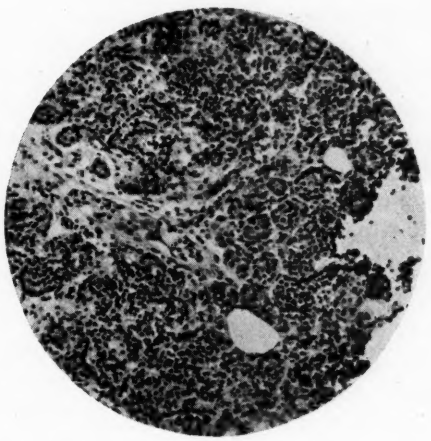


FIG. 299.—*Case* 3. Microphotograph of section of the parathyroid tumour (hæmatoxylin and eosin).

in ill-formed vesicles (*Fig.* 299). This is reminiscent of the appearance sometimes seen in fœtal adenoma of the thyroid gland. A very few oxyphile cells are present.

It will be remembered that in this patient the bone changes were more advanced and more widespread than in the other two, although the blood calcium at the time of operation was very little raised. Our knowledge is not yet sufficient to enable us to say whether the vesicular or non-vesicular appearance is associated with differences in the functional activity of the tumour. Over small areas the appearance of the parenchyma is similar to that of the other two tumours.

DIAGNOSIS.

An association of the generalized form of osteitis fibrosa with hyperparathyroidism has been shown sufficiently often to suggest that hyperparathyroidism may prove to be the cause of generalized osteitis fibrosa. If this should be so, then a diagnosis of generalized osteitis fibrosa would be sufficient to justify an operation on the parathyroid glands. In the present state of knowledge this attitude is not justifiable, and the demonstration of the presence of hyperparathyroidism should be made before operative treatment is undertaken. In each of the three cases reported here, the presence of

generalized osteitis fibrosa and also of hyperparathyroidism was demonstrated, but the grounds for the diagnosis differed in the three cases. In none of the cases was there doubt that the patient was suffering from generalized osteitis fibrosa. All had pains and aches in the limbs, spontaneous fractures, cyst-like areas of rarefaction in the bones, general rarefaction of the bones elsewhere, muscular weakness, and wasting. In one (*Case* 3), there were cellular tumours of bone, and in each of the three cases the cysts or tumours caused expansions of the bones sufficient to be recognized by clinical examination. Renal calculi were present in two of the cases.

In *Case* 2 the evidence of hyperparathyroidism was complete (Barr and Bulger (1929), Bulger, Dixon, and Barr (1930)). The serum calcium was constantly abnormally high, the serum inorganic phosphorus abnormally low, the output of calcium exceeded the intake even on a generous diet, and the urinary excretion of calcium was especially increased. In *Case* 1 the evidence was nearly as good, for the serum calcium was constantly high, and the serum phosphorus low, but calcium was being stored when the intake was adequate. The high urinary excretion of calcium was, however, so striking a feature even on a low calcium intake that the presence of hyperparathyroidism was diagnosed with confidence. In *Case* 3 the evidence was less clear. On one occasion the serum calcium was abnormally high; but during the periods of the controlled observations it was not found to be above a high normal level, although the serum phosphorus was consistently low. The balance experiments failed to show an excess of calcium output over intake except when the intake was markedly inadequate. The high urinary excretion of calcium, however, on a diet containing an inadequate amount of calcium, though not so striking as in *Case* 2, seemed, in conjunction with the other evidence, sufficient to warrant an operation on the parathyroid glands.

It is clear that difficulties may arise in the diagnosis of hyperparathyroidism unless the serum calcium is abnormally high. In *Case* 1, five years before the patient was finally treated by operation, and at a time when the generalized osteitis fibrosa was undoubtedly present, the serum calcium was normal. In *Case* 3 only one estimation out of four showed a definitely abnormally high level of the serum calcium. In such instances elaborate investigations of calcium metabolism are necessary to establish the diagnosis, and even then the faulty metabolism may not be detected unless low calcium intakes are utilized and the high urinary excretion is demonstrated. These findings suggest that even in the presence of a parathyroid tumour, the excessive activity of the parathyroid glands will vary greatly from time to time, so that investigations at any one period in the disease may fail to demonstrate the presence of hyperparathyroidism. In the three cases reported here the most constant abnormality was the increased urinary excretion of calcium.

The relation between the disease of the bones and the hyperparathyroidism is not clear, but whether the disease of the bones is secondary or primary, it would appear that the lesions in the bones will be the immediate cause of the recognition of the condition. The diagnosis of

generalized osteitis fibrosa is not difficult when the lesions are advanced, as in most of the cases hitherto reported and in our three cases, but when the lesions are less pronounced, the diagnosis must be more difficult. This difficulty is important, since we have seen that even in an advanced and typical case of generalized osteitis fibrosa with a parathyroid tumour (*Case* 3), the chemical investigations may fail to show clearly the presence of hyperparathyroidism. In the case of a fourth patient an operation was performed on the neck, but no parathyroid tumour or enlargement was found. In this case the evidence for a diagnosis of generalized osteitis fibrosa was not complete, and the chemical investigations were not indicative of hyperparathyroidism, but the possibility that it was an early case was considered sufficient to justify the exploration. The patient was a married woman, 26 years old. At the age of 9 a fracture of the left femur resulted from

FIG. 300.—Skull from case of multiple cystic disease of bones without hyperparathyroidism, showing localized thickening and irregular mottled appearance about junction of occipital and posterior parietal regions.

a trivial fall, and she had complained of pains in the left leg off and on since the accident. When admitted to hospital in December, 1930, the femora were both bowed and there was a swelling in the middle of the shaft on the left side. X-ray examination showed :—

1. *Skull* (*Fig.* 300).—Thickness of calvarium normal except for the presence of localized superficial thickening, which was most marked about the junction of the occipital and posterior parietal regions ; irregularly mottled in appearance.

2. *Left Humerus* (*Fig.* 301).—Expansion of central part of shaft with appearance as of cysts with sclerosed bone forming their walls, but cavities not so clear as in true cysts ; cortical layer on inner side of expansion thinner than in control, and bone beneath rarefied ; cortical layer on outer side thickened.

3. *Pelvis.*—Extensive rarefied areas in iliac bones, with appearance as of cysts with sclerosed margins.

4. *Left Femur.*—Expansion of neck and most of shaft; upper part of neck fractured, but arrangement of bone abnormal; lower part of neck occupied by rarefied area like a cyst and expanding bone; shaft curves outwards; cortical layer on inner and outer margins thinner than normal, and outer layer showing wavy outline towards centre of bone; large cavity, apparently cystic, occupying middle of shaft with split in external cortical layer opposite to it; similar cavity lower in shaft and several smaller ones in lower end of bone; whole medulla irregular in appearance as if occupied by less clearly defined spaces.

A B

FIG. 301.—Left humerus from a case of multiple cystic disease of bones without hyperparathyroidism (A)—with a control (B)—showing expansion of central part of shaft with appearance as of cysts with sclerosed bone forming their walls.

5. *Left Fibula.*—Expansion in middle of shaft due to fusiform expansion of medullary cavity with sharply defined upper and lower margins; cortex as thick as in control but density less.

6. Lumbar spine, bones of forearms, tibiæ, right humerus, right femur, and left fibula show no abnormalities when compared with controls.

The left femur was explored in two places. In the central part of the shaft at the site of the old fractures, large intercommunicating cavities were found surrounded by dense fibrous tissue. In the neck of the bone, at the site of what appeared to be a large cyst, there was no cavity, the area being filled with masses of firm fibrous tissue. The fibrous tissue from the two places was similar, and on microscopical examination showed no normal bone or marrow, but consisted of dense fibrous tissue well formed and very vascular. Embedded in this were many small pieces of bone, irregular in shape and consisting of woven bone without a proper lamellar structure. There was no osteoid layer and no evidence either of resorption by osteoclasts or of apposition.

The chemical investigations showed normal values for the calcium and phosphorus in the serum, and no abnormality of calcium and phosphorus metabolism was detected except that the urinary excretion of calcium was slightly higher than that in normal controls, when on a diet providing a low calcium intake. In view of the failure to discover any abnormality in the

parathyroid glands at operation, this case may be one of focal osteitis fibrosa, but it is right to point out that the criteria, recognized at present, for a diagnosis of generalized osteitis fibrosa are based on the study of advanced cases, and that studies at different stages, and especially in the early stages of the disease, not only of the bones but also of the parathyroid glands, are lacking.*

From the observations on our three cases and the reports in the literature of other cases, generalized osteitis fibrosa can usually be distinguished from focal osteitis fibrosa on the following grounds. In either disease any bone may be affected; the general affection of the calvarium seen in the cases with hyperparathyroidism has not been recorded in focal osteitis fibrosa; the changes that have been recorded in the latter disease, as in the case without parathyroid tumour described above, differ greatly, as seen in skiagrams, from the changes seen in generalized osteitis fibrosa. Osteoporosis of the bones generally, and particularly in parts which do not show cystic changes, is characteristic of generalized osteitis fibrosa, and can usually be recognized by a diminution in the thickness of the cortical tissue or by an increase in the size of the Haversian spaces, but is best demonstrated by means of X rays by comparison with controls. The osteoporosis is not always very evident in the early stages of the disease (*Case* 2), but is at no time evident in focal osteitis fibrosa. As a rule thickening of bone is seen only in the calvarium in cases of generalized osteitis fibrosa, but was present in other bones in *Case* 3; in focal osteitis fibrosa the bones are usually much thickened at the site of the massive fibrosis of the marrow. Skiagrams in generalized osteitis fibrosa fail to show fibrosis of the marrow, as it is a fine fibrosis, differing from the massive fibrosis of focal osteitis fibrosa, which replaces the medullary cavity and may be recognized in skiagrams by reason of the fact that the outlines of the fibrotic mass throw visible shadows.

If the bony lesions are explored, further evidence of value in differentiation is obtained. True cysts are unusual in focal osteitis fibrosa, but have been reported; in generalized osteitis fibrosa they are usual, and may be central, expanding the bone, or may be in the cortical layer. The cyst-like appearances seen in the focal disease are due to the replacement of the bony structures by fibrous tissue. Osteoclastomata occur as gross tumours in a comparatively small proportion of cases of generalized osteitis fibrosa. In the cases with the lesions of fibrocystic disease in a number of bones but without hyperparathyroidism, osteoclastomata have not so far been found; in localized fibrocystic disease every stage may be found from a typical

* Since the preparation of this article, a very early case under the care of A. F. Hurst and J. F. Venables has been operated upon and a tumour removed by one of us (T. P. D.). This case was orally reported at the meeting of the Association of Surgeons in May, 1932, and will be reported elsewhere. In this patient neither deformity nor fracture had occurred. Great muscular weakness associated with aching pains—especially in one hip region—led to an X-ray examination. Decalcification of bones appeared to be present. This was confirmed by taking controlled skiagrams of a number of long bones. Examination of the blood serum revealed a high calcium content. A parathyroid tumour was found in the middle of the right lobe of the thyroid. The pains disappeared and muscular strength returned following the operation.

osteoclastoma, through cysts with giant-cell tissue in the walls, to cysts in which only a few osteoclasts are found in the margin.

Apart from a study of the bones there are other criteria of value in the differentiation. Generalized osteitis fibrosa is twice as common in women as in men; the onset is usually in adult life or in adolescence; bone pain is usual apart from or previous to the occurrence of fracture; bending of bones occurs from a fracture or collapse of a cystic area; muscular weakness and hypotonicity are common; renal calculi are common; and when the onset of the condition occurs in young subjects there may be delayed general and sexual development. Focal osteitis fibrosa seems to occur equally in males and females; the onset is usually in childhood or adolescence; bone pain is absent or slight except when fracture occurs; bending of bone occurs particularly in the upper ends of the femora and in the tibiæ; muscular weakness, wasting, delayed sexual development, and renal calculi, do not occur.

SUMMARY.

1. Three cases of generalized osteitis fibrosa with hyperparathyroidism are described, with an account of the bony lesions as determined by exploratory operations and X-ray examinations, and the results of chemical investigation of the calcium and phosphorus metabolism.

2. In each of the three cases a tumour of the parathyroid glands was removed by operation, and in each case the situation of the tumour was unusual. Tetany resulted from the operation in two of the cases. A brief description is given of the naked-eye and histological characteristics of the tumours.

3. The diagnosis of generalized osteitis fibrosa depends on the demonstration of generalized rarefaction of the bones, in addition to the presence of cyst-like areas of rarefaction, cellular tumours of bone, spontaneous fractures, muscular weakness and wasting, and aches and pains in the limbs. Renal calculi were shown to be present in two of the three cases on X-ray examination.

4. An abnormally high level of serum calcium was not always found, even when a parathyroid tumour was present. In these three cases an increased excretion of calcium in the urine was constantly demonstrated.

5. In cases of generalized osteitis fibrosa the presence of hyperparathyroidism should be demonstrated, before operation on the parathyroid glands is undertaken.

6. After the removal of the parathyroid tumour the level of the serum calcium fell strikingly, and calcium was retained by the body, or, if retention was present previously, this was definitely increased.

7. An abnormal condition of the phosphorus metabolism was found in each of the three cases, but no consistent relation could be demonstrated between the changes in the calcium metabolism and in the phosphorus metabolism, either before or after the removal of the parathyroid tumours.

8. As a result of the removal of the parathyroid tumour, striking improvement in general health and in strength, and cessation of pain, resulted

in each of the three cases, and no further fractures occurred. A considerable increase in the density of the bones, especially around the cysts and in the cortical bone, was seen from ten to twenty-four months after the operation. In *Case* 2 the appearance of the skull showed a striking return towards the normal twenty months after the operation.

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CHRONIC PAROTITIS: A REPORT OF FOUR CASES WITH SIALOGRAMS.

By LESLIE N. PYRAH,
SENIOR SURGICAL TUTOR, UNIVERSITY OF LEEDS.

Chronic infections of the parotid gland are sufficiently uncommon to arouse special attention, and four such cases are recorded below. All four cases have been investigated by sialography, which consists of a radiographic study of the architecture of the larger and smaller ducts of the gland by the injection of radio-opaque fluids into Stenson's ducts; lipiodol is the fluid selected for injection.

Fig. 302.—Sialogram of normal parotid gland.

The technique of sialography has recently been described by Payne,[1] who also referred to the somewhat scanty literature upon the subject, and by Pyrah and Allison[2]; the papers of these writers should be read for further details. It suffices here to say that ½ to 1 c.c. of lipiodol is injected into Stenson's duct and a lateral radiogram is immediately taken, the head of the patient being fully extended. A normal sialogram is reproduced for purposes of comparison (*Fig.* 302).

CASE REPORTS.

Case 1.—E. V. J., male, age 36.

History.—The patient stated that in April, 1931, he suffered from an attack of influenza and immediately afterwards had a swelling of the left parotid gland. The swelling was marked during and after meals and subsided two or three hours later; when the gland was swollen he experienced some discomfort but no actual pain. During the acute stage he did not notice any discharge into the mouth. For some weeks later the swelling persisted, though it became noticeably less; but even in three or four months' time there was still a slight swelling of the gland after meals, and he then began to notice a salty taste in his mouth. Pressure over the gland caused a discharge of saliva which had a salty taste.

On Examination (Jan. 1, 1932).—There was a slight enlargement of the left parotid gland, especially of the socia parotidis. The surface of the enlarged gland was quite smooth and there was definite tenderness on pressure. The orifice of the left Stenson's duct was slightly injected, and the saliva which was squeezed out on pressure over the gland was slightly purulent. The right parotid gland was normal. The teeth were healthy, radiological examination showing no apical disease, and the throat and nose (which were examined by Mr. E. W. Bain) were also found to be healthy. The Wassermann reaction was negative. No organisms were grown from the saliva, but pus cells were present. A sialogram of the left parotid (*Fig.* 303) showed that Stenson's duct was dilated to about four times its normal calibre but that there was no abnormality of the smaller ducts. A sialogram of the normal right parotid gland showed an exactly similar picture. Calculi in the gland were excluded by preliminary X-ray examination.

Fig. 303.—*Case* 1. Sialogram of left parotid gland. This shows an enormous dilatation of Stenson's duct and of the terminal portion of its chief contributory ducts.

Treatment.—Treatment by light massage to the gland, acid drinks to promote the flow of saliva, and mouth-washes gave no relief. Though no gross obstruction of the orifice of the gland was demonstrated, it was thought that the dilated duct resulted in some kinking at its orifice in the mouth. The terminal portion of the duct was therefore slit up for half an inch under local anæsthesia and weekly injections of lipiodol were given along the duct. The saliva is now normal and there is no salty taste in the mouth; there is still, however, occasional fullness in the gland after meals, but the condition is much improved.

Case 2.—E. W., female, age 37.

History.—The patient stated that since July, 1931, she had had a sore mouth, particularly marked on the inside of the cheeks. A few weeks later she noticed a salty taste in the mouth and a swelling of both parotid glands. There was no appreciable increase in the size of the glands after food, but it was often considerably increased in cold weather. In December, 1931, all her teeth had been extracted without any improvement in the condition. Examination of the nose and throat revealed nothing abnormal. Since January, 1932, the patient has had a very dry mouth.

On Examination (Jan. 30, 1932).—Both parotid glands were slightly enlarged and tender. The orifices of both Stenson's ducts in the mouth were very red, œdematous, and pouting. The mucous membrane of the cheek surrounding the orifices of Stenson's ducts was the seat of an angry-looking granular stomatitis, associated in places with deep furrows evidently due to submucous fibrosis. The mucosa also showed yellowish-white areas of fibrosis (*Fig.* 304). The tongue was normal. There was a discharge of muco-pus from both parotid glands upon pressure over them. The Wassermann reaction was negative. Sialograms of both parotid glands showed very slight but definite dilatation of the smallest ducts (*Fig.* 305).

Treatment.—The chronic stomatitis improved very considerably by treatment with glycerinum boracis and hydrogen peroxide mouth-washes. The flow of saliva was promoted by regular massage to the gland and by acid drinks, and the general

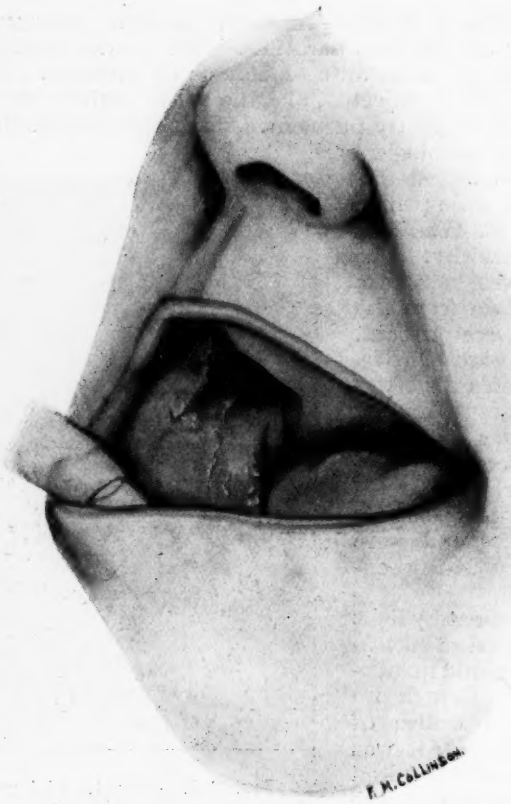

FIG. 304.—*Case* 2. Appearance of mouth. There is an area of chronic granular stomatitis around the orifice of the right Stenson's duct, with much submucous fibrosis.

FIG. 305.—*Case* 2. Sialogram of right parotid gland. There is slight dilatation of the smallest ductules.

condition of the patient was improved by tonics and cod-liver oil. Weekly injections of lipiodol along Stenson's duct were given. The stomatitis almost disappeared, leaving only the furrows and fibrosis mentioned above, and the discharge of purulent saliva has now completely stopped.

Case 3.—O. B. S., female, age 47.

HISTORY.—The patient had an attack of acute right parotitis in April, 1918. The swelling, which was considerable, subsided in about a fortnight, but a few weeks later she noticed a discharge into the mouth of thick purulent mucus, coming, apparently, from the right parotid gland; this discharge ceased in a few weeks' time. In 1927, following an attack of influenza in which the tonsils were somewhat swollen, the right parotid gland again became slightly swollen and painful. In 1930 the patient had a third attack of acute parotitis of the right gland, followed a fortnight later by a similar change in the left parotid gland; these attacks were associated with swelling, considerable pain, trismus, and with slight elevation of temperature. After this third attack the patient complained of very marked dryness of the mouth and a mucopurulent discharge from both parotid glands; these symptoms have persisted ever since. The patient has had her tonsils enucleated; they were heavily infected with a hæmolytic streptococcus. She has had prolonged treatment with vaccines prepared from cultures from the tonsils and from the parotid (which also yielded a hæmolytic streptococcus). She also had the right antrum washed out. Stenson's duct has been repeatedly irrigated with a weak solution of iodine.

FIG. 306.—*Case* 3. Sialogram of right parotid gland. Shows an incomplete filling of the ducts due to fibrosis-atrophy of the gland.

ON EXAMINATION (February, 1932).—The parotid glands were not enlarged. On pressure over the right gland and to a less extent over the left a small amount of bubbly muco-pus could be expressed; microscopic examination of the discharge shows the presence of pus cells. The orifices of Stenson's ducts in the mouth are prominent and slightly red. There are no foci of infection in the teeth or in the mouth. A sialogram shows incomplete filling of the smaller ducts of the parotid evidently due to advanced fibrosis (*Fig.* 306).

TREATMENT.—The patient could not tolerate acid drinks owing to the extremely dry mouth, but evacuation of the ducts has been promoted by massage to the glands. A prolonged course of lipiodol injections, at first at weekly intervals and later every two or three days, has been given, but no improvement has taken place.

Case 4.—M. H., female, age 16. This case has been reported elsewhere, but is recorded here again to complete this series.

HISTORY.—The patient first came to the Leeds General Infirmary early in 1929 with a discharge of pus from the left ear, and returned a few months later with a swelling of the left parotid gland, which was followed a few weeks afterwards by a swelling of the right gland. At first the glands were but slightly swollen, but in July the right parotid became acutely painful, hot, and swollen; similar acute changes occurred in the left gland a few days later. There was no elevation of temperature. The inflammation in both glands subsided in a few days after

treatment by fomentations and hot mouth-washes. During the following five years both glands have been persistently larger than normal and there have been acute exacerbations every few months. The patient can at any time squeeze from the glands a considerable amount of ropy muco-pus, which, especially during the acute crises, has a very offensive taste.

On Examination (January, 1930).—Both parotid glands were enlarged and their surfaces were lobulated throughout; there were no signs of inflammation, but the glands had a fibrous consistency. On pressure over a gland a mixture of clear saliva and ropy mucus could be squeezed from Stenson's duct, the orifice of which was very prominent and slightly injected. On examination during an acute attack some weeks later the glands were considerably swollen and their surfaces were smooth. By gentle massage of the gland saliva containing plugs of pus mixed with epithelial débris could be squeezed out of Stenson's ducts in considerable quantity. The acute attacks lasted from one to three weeks. The teeth were very unhealthy and showed a deficiency of enamel, many having broken off near the alveolar margins while all the remainder were carious. The throat and tonsils were healthy; the discharge from the ear subsided three years ago. The blood-count was normal and the Wassermann reaction was negative. X rays revealed no parotid calculus. A culture of the débris discharged from the duct gave a growth of a green-forming streptococcus; the débris itself consisted of pus cells in large quantity, mucus, and desquamated epithelial cells. Sialograms of both glands showed a very marked dilatation of the terminal alveoli; the extent of this dilatation was uniform throughout the gland, and the picture resembles that of bronchiectasis of the lungs (*Fig.* 307).

Fig. 307.—*Case* 4. Sialogram of right parotid gland. Shows enormous dilatation of the smallest ductules.

Treatment.—All the teeth have been removed. The condition is showing gradual and marked improvement after prolonged massage to the glands and the use of acid drinks and mouth-washes and occasional injections of lipiodol into Stenson's duct. The girl's general condition has been considerably improved by artificial sunlight, tonics, malt extracts, and by short periods of convalescence in the country; she now appears to be progressing towards a complete recovery.

COMMENTARY.

Pathology.—If the specific chronic infective diseases of the parotid gland are excluded—namely, syphilis, tuberculosis (including uveo-parotitic fever), actinomycosis, and the condition known as Mikulicz's disease—there remain a number of non-specific infections of the gland which have probably a varied etiology.

Cases 2, 3, and 4 above are examples of chronic parotitis arising from infective foci in the mouth. In *Case* 2 a chronic stomatitis had preceded the

parotitis and the two conditions subsided synchronously after treatment. *Case* 3 had had an infection of the tonsils (by a hæmolytic streptococcus) immediately preceding the initial acute attack of parotitis, while in *Case* 4 the parotid infection probably arose from carious teeth.

In *Case* 1 the parotitis possibly resulted from a blood-borne infection, but it is equally possible that the infection ascended Stenson's duct from the mouth, which, during the febrile stages of the influenzal attack, is dry and likely to be harbouring pathogenic organisms. The great dilatation of Stenson's duct is of interest in this case; as it is bilateral it is probably congenital. The case report indicates that there seemed to be a definite obstruction near the orifice of the duct, the lumen of which is quite four times that of a normal Stenson's duct; once free drainage of the duct had been provided, the infection rapidly disappeared. A similar case of great dilatation of one Stenson's duct was reported by Barsony.[3] Such a condition must be very rare.

There is no doubt that in some cases of chronic swelling of the parotid gland the infective lesion lies in the ducts. Raymond Johnson[4] reported such a series. The patients had a unilateral painful enlargement of the parotid gland which became larger after food. Pressure over the gland caused the extrusion of a plug of mucus from the duct followed by a flow of watery saliva; this gave a temporary relief of symptoms. The orifice of Stenson's duct in the mouth was red and inflamed. The swellings usually disappeared after some months of treatment, but occasionally they reappeared at intervals.

Kussmaul[5] described some cases of chronic parotitis in adults which he called 'sialodochitis fibrinosa'. In half of his cases the condition was bilateral. The parotid gland became suddenly swollen and the patient had considerable pain, trismus, and in some cases fever. The condition was relieved after some hours, or days, by the extrusion from Stenson's duct of a long plug of fibrin; following the fibrin, a stream of saliva escaped and the swelling of the gland subsided. Kussmaul compared these cases with bronchitis fibrinosa.

No biopsies have been possible in any of the four cases reported, but the sialograms throw some light upon the pathology. The normal architecture of the ducts of the parotid gland is shown by the normal sialogram, which is a typical one taken from a number of normal ones. The sialogram of *Case* 2 shows the earliest deviation from the normal picture; here Stenson's duct is of normal size, but many of the terminal ductules show small dilatations while others are perfectly normal; the disease in this case subsided in about nine months.

The sialogram from *Case* 4 shows a much later stage in the disease; here, after four and a half years of chronic infection, the terminal ductules are so dilated that they appear on the sialogram as spheroidal spaces and they resemble perfectly the dilatation of the terminal bronchioles in bronchiectasis of the lungs. The large dilatations of the terminal ducts allow of the accumulation of much mucopurulent débris, and, while they are probably the result of infection, they also serve to perpetuate it indefinitely. Both Keith[6] and Payne[1] have recorded sialograms of similar cases.

The sialogram of *Case* 3 shows quite a different result of chronic long-standing infection. Although exactly the same syringe and technique were

employed as for the other sialograms, the filling of the terminal ductules was for the most part incomplete, though here and there very small dilatations can be seen; the gland in this case has evidently undergone very advanced fibrosis as a result of the long-standing infection, and this fibrosis is reflected clinically in the extreme dryness of the mouth from which the patient suffers.

Calculi were present in the parotid glands of none of the four cases. Though uncommon, calculi do accompany a number of cases of chronic parotitis; a recent series has been recorded by New and Harper.[7]

Symptomatology.—All four cases have had an acute or subacute onset, but none has proceeded to the stage of suppuration. During the acute stage the gland has been hot, tender, and painful. One case (*Case* 4) has had repeated subacute exacerbations which mimicked the first attack. Similar cases of recurrent parotitis have been described by Walker,[8] Payne,[1] and by Hobbs, Sneierson, and Faust.[9] During the chronic phase of the disease there was either no discomfort at all or a little fullness of the gland after meals. Throughout the disease pus in smaller or larger amounts is discharged from Stenson's duct.

It is of interest here to note the occasional occurrence of aerocele of the parotid gland, and a case has very recently come to my notice; such a condition may mimic subacute infections. The patient, a girl of 10, habitually had a glass of milk in the evenings, and sometimes she took the milk from the glass with an exaggerated sucking action of the mouth. On several such occasions the parents noticed an acute enlargement of both parotids which rapidly diminished upon cessation of drinking and upon gentle massage of the gland. On one occasion the father, who is a medical man, noticed that synchronously with the swelling of both parotids an extensive surgical emphysema of the cheek and side of the head had developed. The provisional diagnosis of aerocele of the parotid gland was thereby confirmed; the emphysema must have developed from a tiny tear in Stenson's duct or in a smaller duct resulting from the pressure of air aspirated into the gland. A similar case of acutely developing parotid swelling in a baby was described by Wolff (cited by Steinitz[10]), and he suggested that the cause of the swelling was a spasm of the orifice of Stenson's duct; the case, however, was probably one of aerocele of the parotid gland.

Treatment.—Rational treatment consists in the treatment or the removal of foci of infection in the mouth, in the promotion of secretion from the gland in order to ensure evacuation of the purulent débris which accumulates in the ducts, and in irrigation of the ducts with some mild non-irritating antiseptic.

The treatment of infective foci in the mouth may be assisted by the administration of vaccines prepared from cultures of the organisms in such foci; in the solitary case in which this method has been tried the patient derived no benefit (*Case* 3). In *Case* 2 the improvement of the parotitis ran parallel to the improvement of the stomatitis.

Promotion of drainage of infective material from the ducts is best obtained by gentle massage to the gland; this can be done, after instruction, by the patient himself, and consists in slow oft-repeated pressure with the

fingers moderately firmly over the gland from behind forwards. The patient can easily perform the treatment several times daily. The flow of saliva can be increased by acid drinks (lemonade, etc.) throughout the day and by the frequent use of chewing gum. Calculi in the gland or in the duct should be removed and cicatricial narrowings in the duct should be dilated or divided. Hobbs, Sneierson, and Faust[9] have carried out dilatation of the orifices of Stenson's duct for a series of cases of acute and chronic parotitis, using filiform bougies. Most cases of acute parotitis subside without such treatment, and it is difficult to see what it can offer in the absence of a stricture of the duct.

The instillation of ½ to 1 c.c. of lipiodol into Stenson's duct has proved successful in two of the cases of parotitis reported above. Lipiodol is non-irritating, contains about 50 per cent of iodine, and, being a highly viscid oil, remains in the ducts for several hours before it is discharged. Payne also has found this method of treatment very useful. The injections are made at weekly intervals, and for short periods injections may be given on alternate days. Hobbs, Sneierson, and Faust[9] used injections of saline and of a solution of 1 per cent mercurochrome.

SUMMARY.

1. Four cases of chronic parotitis are reported, and the sialograms of these cases are shown.

2. The pathology of the cases is discussed, and the uses of sialograms in elucidating the morbid anatomy of the infected glands and of their ducts are demonstrated.

3. The treatment of chronic parotitis is outlined.

I desire to thank the members of the Honorary Surgical Staff of the Leeds General Infirmary for permission to record these cases which have been under their care.

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THE TECHNIQUE OF RESECTION OF THE PRESACRAL NERVE (COTTE'S OPERATION).

By A. A. DAVIS, Manchester,
DICKENSON RESEARCH SCHOLAR IN SURGERY.

Since Cotte, of Lyons, first described his operation in 1925, in his now classical article in the *Journal de Chirurgie*,[1] the procedure has been widely adopted, more especially in the Latin countries, for the relief of a variety of painful pelvic conditions, particularly gynæcological. Aievoli,[2] Aubert,[3] Bernard and Theodoresco,[4] Chianello,[5] Cotte,[6, 7, 8] Derom,[9] Ferey,[10, 11] Georgesco,[12] Gomoiu,[13] de Grisogono,[14] Hallopeau,[15] Heitz,[16] Learmonth and Braasch,[17] Mornard,[18] Michon,[19] Paolucci,[20] Petersen-Ekkert,[21] Spirito,[22, 23] Tirelli,[24] Viannay,[25] and Walther[26] have all reported good results, and although a certain amount of exaggerated enthusiasm has undoubtedly been exhibited by its more ardent advocates, there is no doubt that the operation is definitely establishing itself as one of the most successful in sympathetic surgery. It is as yet too early adequately to assess the results in the twenty cases operated upon, with Dr. W. R. Addis, during the past year at the St. Mary's Hospital, Manchester, but in view of the rapidly increasing popularity of the operation among English surgeons, it is suggested that a description of certain technical details and difficulties learned in its performance might be of service. (It is of course desirable, here as elsewhere, that cadaveric experiment should precede any actual operation.)

TECHNIQUE OF THE OPERATION.

Continental opinion is divided between Pfannensteil's transverse suprapubic incision and a median subumbilical one, with a bias in favour of the latter. We ourselves employ a left paramedian incision, sliding the rectus outwards. A length of from 3 to 4 in. from the umbilicus downwards is usually adequate, though in fat patients this may have to be prolonged upwards. Here, as elsewhere, æsthetically designed incisions occasionally contrive to produce a certain amount of internal difficulty; and as in these cases one is working at considerable depth, intestinal trauma can be avoided only by a reasonable exposure.

The peritoneum opened, the abdominal and pelvic organs are rapidly examined, and any abnormality is dealt with. (We have never considered it justifiable to omit the surgical treatment of a pelvic organ found requiring it, as advocated by certain French writers, in order to assess the value of presacral resection alone.) This done, the Trendelenburg position is adopted, and the site of operation exposed by careful packing off of the bowel on all sides. This is best accomplished with a single roll of 8-in. gauze, relaxation being much facilitated by the employment of spinal anæsthesia.

The 'three points of Cotte' should next be definitely identified by palpation. They are: (1) The promontory of the sacrum; (2) The bifurcation of the aorta; (3) The inferior mesenteric artery. In this connection it should be remembered that the 'presacral nerve' of Latarjet is neither presacral, nor is it usually a single nerve. In the majority of cases it takes the form of several communicating filaments lying on the anterior surface

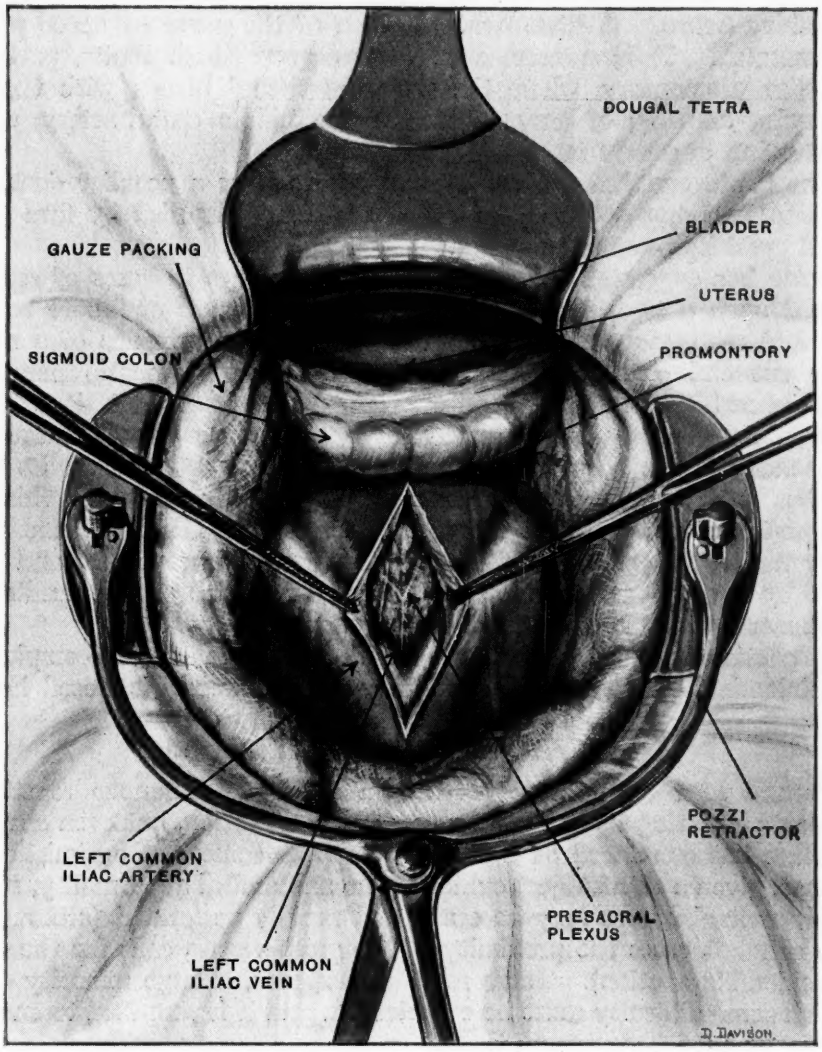

FIG. 308.—Resection of the presacral nerve: the operative field.

of the 5th lumbar vertebra, and must be sought for in this position. The nerves can occasionally be seen in thin subjects, shining through the peritoneum, and can usually be palpated through it.

The posterior parietal peritoneum over the nerves is lifted gently and snipped with scissors, which continue the incision upwards and downwards for an inch or more, exposing the operative field as depicted in *Fig.* 308.

No attempt should be made to dissect out and excise the individual nerves, as this is usually impossible, and is in any case anatomically inadequate. Instead, the layer of fibrocellular tissue in which the nerves run, and which lies between the posterior parietal peritoneum anteriorly, and the left common iliac vein and 5th lumbar vertebra posteriorly, should be systematically removed in one sheet, from the left common iliac vein to the right common iliac artery laterally, and from the aortic bifurcation above to the sacral promontory below. A little over one inch of the nerve or nerve plexus is thus exsected. It is unnecessary to transgress these limits, particularly below the promontory, where the 'carrying tissue' blends with the sacral periosteum, allowing of easy rupture of the middle sacral artery, which in this situation is nearly inaccessible to ligature.

The fascia containing the presacral nerves is of a peculiar density and toughness, and any doubt of its validity is easily dispelled by firm traction exerted upon its proximal cut end.

Cotte has suggested, on the basis of his findings in cases of recurrence operated upon a second time, that an occasional cause of failure to relieve or prevent reappearance of the pain is probably the presence of a neuroma at the cut end of the nerve, analogous with the traumatic neuroma of the peripheral spinal nerves, and secondary to ligature irritation. Leriche and Fontaine[27] have demonstrated their occurrence experimentally, and Bewerschenko[28] has suggested a variety of heroic treatments for its prevention, but probably the most reasonable way of accomplishing this is the *entire omission of ligature* in separating the resected nerve. Hæmorrhage is usually negligible, coming solely from the tiny vasa nervorum, and is controlled by tampon pressure. The occasionally brisk arterial bleeding from an abnormal vessel is easily seen and clipped.

A continuous peritoneal suture of No. 90 linen thread completes the intra-abdominal portion of the operation, and the wound is closed in layers.

DIFFICULTIES AND DANGERS.

Perusal of the literature reveals an almost monotonous repetition of statements declaring the ease and safety of the operation, but even our limited experience has convinced us that these must be somewhat optimistic. It is true that, given a thin subject exhibiting no anatomical abnormality, resection of the presacral nerves may be easily and rapidly performed (although even in the simplest cases the proximity of the great vessels calls for the exercise of considerable caution). There remains, however, a large minority of cases in which some difficulty must be experienced, and it is proposed to enumerate these as we have encountered them.

1. The presence of much fat in the abdominal wall may so increase the depth of the operative field as to make manipulation very difficult. An adequate incision, combined with the highest Trendelenburg position, facilitates matters.

2. The presence of a pelvic tumour may entirely negative the procedure. If simple, it should be removed as a preliminary. If inoperable, resection of the intermesenteric nerves, as suggested by Bernard and Theodoresco[4], may be performed.

3. The pelvic mesocolon, normally confined to the left of the mid-line, may transgress it, thus lying across the field of operation. Roussel[29] found this abnormality in 15 per cent of his anatomical subjects, but this incidence is probably much too high, and in either case its presence is not, as Roussel suggests, a complete bar to presacral resection. Where this difficulty is encountered, it is necessary to incise both leaves of the mesosigmoid in order to expose the peritoneum covering the nerves. The mesosigmoid should be traversed parallel with its root, and half an inch from it, to avoid the superior hæmorrhoidal artery. Dissection between the layers should be carefully avoided, as it confuses localization.

4. We have seen an accessory ureter running in the mid-line on three occasions, closely simulating a single large presacral nerve. It may be identified by palpation and the effect of stimulation.

5. In a case of abnormally low bifurcation of the aorta the inferior mesenteric artery was found running vertically downwards in the mid-line. The nerves in this case were resected piecemeal on either side of the artery.

6. Hæmorrhage from the middle sacral artery is the result of a too zealous denudation of the fibrous tissue covering it as it lies on the 5th lumbar vertebra. It is unnecessary to remove this fascia, from which the superjacent nerve-bearing tissue is easily and definitely separable by blunt dissection. Under no circumstances should the resection transgress the promontory, as rupture of the vessels is then almost inevitable, and subsequent hæmostasis extremely difficult; it is best accomplished by under-running.

7. In a case of pelvic neuralgia secondary to inoperable carcinoma of the cervix uteri (operated upon by Professor Dougal) a single nerve, much enlarged by malignant infiltration, was found only after a long search, adherent to the elevated parietal peritoneum. This occasional cause of difficulty is best surmounted by the routine preliminary stripping of the under surface of this peritoneum, as suggested and employed by Addis, of Manchester.

8. Lastly, extreme gentleness is necessary in stripping the left common iliac vein if damage to its very friable walls is to be avoided. The hæmorrhage caused in this way in one of our cases was fairly easily controlled by lateral ligature.

SUMMARY.

The technique of the operation of resection of the 'presacral nerve' is described. It is usually easy and safe in execution, but the presence of occasional abnormalities calls for the exercise of considerable caution in its performance. A description of these difficulties, as they have been observed in twenty consecutive cases, is given, together with suggestions concerning their treatment.

I am much indebted to Dr. W. R. Addis for his kind help and advice, and to Professor D. Dougal for permission to report his case.

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SHORT NOTES OF RARE OR OBSCURE CASES

DIVERTICULUM OF CÆCUM.

By R. KENNON, Liverpool.

Mr. E., aged 26, was seen on the third day of an attack of pain, tenderness, and rigidity in the right iliac fossa, with slight temperature. A retrocæcal appendicitis was diagnosed.

At operation an inflammatory mass was discovered behind the cæcum 3 in. by 2 in. in extent. On turning the cæcum inward a pale blue flaccid

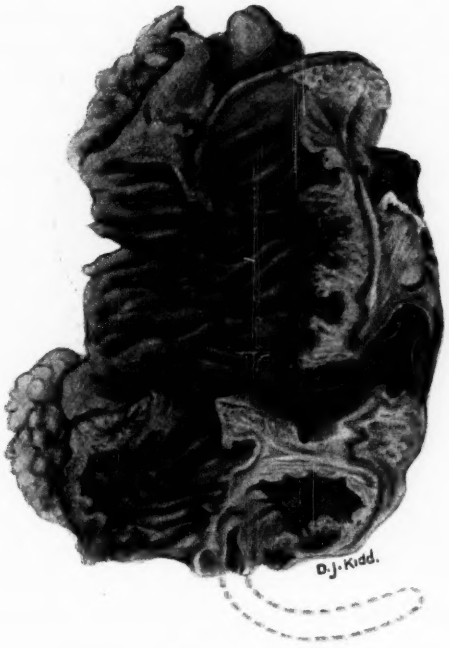

Fig. 309.—Diverticulum of cæcum.

appendix presented itself, obviously normal. This was removed. On further palpation of the retrocæcal mass, the finger entered what was considered to be an ulcer crater $\frac{1}{2}$ in. in diameter with edges heaped up and $\frac{3}{4}$ in. broad. Under the mistaken diagnosis of tuberculosis or malignant disease the ileo-cæcal angle was resected.

Professor Dible, who examined the specimen (*Fig.* 309), reported "A very beautiful diverticulum. The muscle is defective and the floor consists

of fibrous tissue. There is a marked inflammatory change involving more especially the narrowed ostium."

The lumen admitted the little finger for 1½ in. The patient was alive and well a year later.

A DOUBLE GALL-BLADDER OPENING BY TWO CYSTIC DUCTS INTO THE COMMON BILE-DUCT.

By R. KENNON, Liverpool.

Mr. R., aged 69, was first operated upon by me in 1930 for acute suppurative cholecystitis. Several large stones were removed and drainage was established. Delirium prior to operation and a known cardiac arrhythmia reduced operative procedure to the minimum. The anomaly was not recognized.

After a year of good health, painless febrile attacks associated with mild icterus supervened. With some hesitation, owing to the condition of the heart, it was decided to explore the common bile-duct in May, 1932. The

Fig. 310.—A double gall-bladder opening by two cystic ducts into the common bile-duct.

wider exposure revealed the presence of two gall-bladders—the upper one, 5 in. long and 1½ in. in diameter, containing a faceted stone; and parallel to it on its lower side, a small gall-bladder with thicker walls, 3 in. long and ¾ in. in diameter, free from stones, but with a scarred fundus, the result of previous operation (*Fig.* 310). Each gall-bladder opened separately into the common bile-duct, which contained several stones and débris.

Cholecystectomy with drainage of the common bile-duct was performed. Auricular fibrillation and a pulmonary embolus complicated a rapid local convalescence. Recovery eventually took place.

A CASE OF FACIAL PARALYSIS TREATED BY FASCIAL GRAFTS.

By R. BROOKE,

ORTHOPÆDIC SURGEON, ROYAL WEST SUSSEX HOSPITAL.

The treatment of permanent facial paralysis due either to acute mastoid disease or to post-operative trauma is a problem which has exercised the ingenuity of the surgeon to the utmost; and it must be admitted that so far no method has been devised which deals with this complication in a satisfactory manner.

Nerve anastomoses are beset with technical difficulties, and even in the hands of the expert these operations on occasions fail. When they are successful the combination of voluntary movements with the movements of the face is extremely irksome to the patient and tends to mitigate and spoil an otherwise perfect functional result.

Section of the cervical sympathetic trunk is useful in dealing with the inflammation of the eye from which these patients so often suffer, by allowing the eyelids to come together when the levator palpebræ superioris is relaxed. It has the disadvantage that it does not overcome the facial asymmetry or improve the appearance of the mouth. In young people, especially young women, this is important, although to the patients themselves the greatest disability, and the thing about which they complain, is not the deformity, but the pain and interference with vision owing to the exposure of the eyeball to the air.

The following case illustrates a method of treating this disability by means of fascial strips which are obtained from the thigh, and are used to draw together the eyelids and elevate the angle of the mouth.

The patient, Mrs. C., aged 48 years, was first seen on Feb. 8, 1932, when she gave a history of complete right-sided facial paralysis following acute mastoid disease three years previously. She complained of pain in the right eye and interference with vision.

Operative Details.—Using a modification of the method described by Passot, strips of fascia lata, approximately $\frac{1}{8}$ in. wide and 12 to 14 in. in length, are obtained from the thigh. A small vertical incision $\frac{1}{4}$ in. in length is then made slightly above and in front of the ear at the level of the commencement of the helix. This incision is situated in the hair-bearing area of the scalp, which is prepared for operation by cutting short the hair in this region in preference to shaving, as the latter may give rise to a local furunculosis.

In the depths of the wound the superficial temporal aponeurosis and the upper margin of the atrophied auricularis anterior muscle are identified. An ordinary malleable metal probe is then taken, and through the flattened eyelet end a fascial strip is threaded. The probe, eyelet end forward, is then introduced into the wound and pushed towards the inner canthus of the eye. No force is required for this, and the probe can be guided quite

readily beneath the skin along the upper eyelid. As it is malleable, it adapts itself in its passage to the conformation of the face.

A small curved incision ¼ in. in length is next made on the side of the nose half way between the inner canthus of the eye and the dorsum nasi, at the level of the inner canthus; and the lateral border of the atrophic and fibrotic procerus muscle identified. It is better to make this incision at the side of the nose rather than near the inner canthus. A wound at the inner canthus is mechanically more difficult to close and more liable to become infected by tears from the eye.

FIG. 311.—Fascial strips passed round eye to approximate eyelids.

The eyelet end of the probe is then delivered through the inner wound, unthreaded, and the end of the fascial strip drawn out. The probe is then withdrawn through the original incision and reintroduced, this time passing to the inner canthus beneath the skin of the lower eyelid near its margin until the eyelet end once more projects through the incision at the inner canthus. Part of the procerus muscle and aponeurosis are then freed from their attachment to the nasal bone, and round this the fascial strip is passed. Passot advises isolating the internal palpebral ligament and passing the fascial strip round this. If this is done, there is a risk of interfering with the tear ducts, and the resulting epiphora greatly increases the risk of infecting the wound.

The strip is then rethreaded on to the probe, and drawn back beneath the skin of the lower eyelid to the original incision. The two ends of the fascia which project from the wound are drawn tight enough to close the eyelids. Part of the fibrotic auricularis anterior and aponeurosis are isolated, and round this strips are passed and tied. (*Fig.* 311.) The incisions are closed with fine interrupted horse-hair stitches.

FIG. 312.—Fascial strips passed to angle of mouth to draw up the depressed corner.

The mouth on the affected side next receives attention. The fascial strip used to elevate the corner of the mouth can be introduced through the original incision made near the front of the ear, but in practice better results are obtained if a separate incision ¼ in. in length is made rather farther forward over the zygomatic process, midway between the outer canthus of the eye and the external auditory meatus. Through this incision the remains

of the zygomaticus are identified. The second incision is horizontal at the level of the angle of the mouth and should be at least $\frac{1}{4}$ in. away from the angle of the mouth, as infection of this incision will readily occur if it is placed too close to the lips. In the depths of the incision the atrophied

FIG. 313.—The patient before operation.

FIGS. 314, 315.—The patient after operation.

risorius muscle will be seen. The fascial strip is passed in this case from the upper incision down to the lower one, round part of the risorius muscle and fascia, and finally back again to the original incision, where the ends are drawn tight and tied round the remains of the zygomaticus muscle, which serves as an anchor. (*Fig.* 312.)

The degree of tension in elevating the mouth should be such that the drooping of the angle is over-corrected, especially if the operation is performed under general anæsthesia. In the case illustrated (*Figs.* 313–315), although at the time of the operation the angle of the mouth appeared to have been raised sufficiently, when the patient had recovered from her anæsthetic it was seen that the elevation was not quite enough. It may be impossible to identify the atrophied facial muscles in the depths of the wound, in which case the fascial strips can be anchored to parts of the aponeurosis.

The advantage of this operation over those previously mentioned for the treatment of facial paralysis lies in its simplicity. It may be performed quite easily with local anæsthesia, and needs no special technique or detailed anatomical knowledge. It is much less dangerous than either of the other operations. Especially does this apply to the operation on the cervical sympathetic trunk, where the proximity of the carotid sheath and the vagus nerve is a potential danger even in the most skilled hands.

The method is open to the criticism levelled at all fascial operations, that in time the fascia will stretch. It can only be said that these fascial strips live, and it would not seem unreasonable to suppose that they retain the properties of the fascial band from which they were obtained. In this instance the operation is of too recent a date for any useful observations, but so far, after three months, there are no signs of any relapse, the patient has lost the pain in the eye, and has noticed a distinct improvement in her vision.

SPONTANEOUS RUPTURE OF THE SPLEEN.

By. J. M. BLACK,

SURGEON, DUNFERMLINE AND WEST FIFE HOSPITAL, DUNFERMLINE.

According to Hamilton Bailey[1] there are only eleven cases of spontaneous rupture of the normal spleen on record, and the following case therefore seems to be worth reporting, if only on account of the rarity of the condition.

The patient, a married woman of 52, was admitted to the Dunfermline and West Fife Hospital on Nov. 18, 1931, with a diagnosis of strangulated left femoral hernia, for immediate operation.

History.—She was too ill on admission to give any history, but had had no vomiting. After recovery she was able to give the following history. For about three years she has had a small swelling in the left groin, and on the night prior to admission she felt pain in the left groin and went to consult her doctor, who told her to rest. For several weeks prior to this she had had an uneasy feeling in the left side just below the heart, and she thought she was suffering from heart disease. She is quite definite that she never had a knock or any other injury to her left side. She was up and doing her housework as usual on Nov. 18, when at mid-day she turned very sick and faint with pain in the left hypochondrium, and collapsed. She had suffered from

no previous illness of note. She has had a family of seven, the eldest being 31 years old. There have been three miscarriages. Menstruation ceased in 1929. The Wassermann reaction was negative.

On Examination.—The patient was a well-nourished woman of the maternal visceroptotic type. She was in an extreme state of collapse, with a subnormal temperature and a pulse of 140 per minute, and was very restless. The abdomen was slightly distended and moved well on respiration. There was tenderness all over the left side of the abdomen, maximum in the left iliac region. There was a small hard nodule in the region of the left femoral canal, not tender, with no impulse on coughing, and dull on percussion. There was dullness in the left flank on percussion.

Diagnosis.—Obviously the patient was suffering from an internal hæmorrhage, and in spite of her age the most likely diagnosis appeared to be ruptured extra-uterine pregnancy.

Operation.—Operation was performed immediately under local infiltration with novocain and adrenalin. A left gridiron incision was made. On

Fig. 316.—Spleen showing ruptured capsule and subcapsular hæmatoma.

opening the peritoneum dark fluid blood exuded in large quantity. The uterus was rapidly palpated and was small and involuted; the ovaries and tubes were normal. The great omentum was normal, no evidence of torsion being found.

Light ether anæsthesia was then begun and the splenic area palpated. Immediately there was a copious flow of blood. The incision was extended upwards paramedially, to the left costal margin, and the splenic pedicle was clamped and the spleen removed (*Fig.* 316). It was lightly adherent to the diaphragm by blood-clot, and the left subphrenic space was full of clot and fluid blood. The splenic pedicle was tied with catgut, and the abdominal incision was closed with through-and-through silkworm gut stitches. Pressoplast lace dressing was applied.

One pint of gum-saline and glucose was given intravenously before the

patient left the table, and two hours after operation 250 c.c. of citrated blood was administered, her husband being found a suitable donor.

She recovered from the operation, and, apart from a slight left-sided pleurisy and an attack of epistaxis, her convalescence was uneventful.

SUBSEQUENT PROGRESS.—A blood-count done on Dec. 9, 1931, showed :—

Red blood-corpuscles	3,600,000
White blood-corpuscles	6000
Differential Count.— 471 cells	
	Per cent
Neutrophils	51·9
Lymphocytes	37·1
Monocytes	7·6
Eosinophils	3·2
Basophils	0·2

Stained film showed some polychromasia.

The stitches were removed on Dec. 3, and the patient was discharged from hospital on Dec. 18, one month from the date of admission.

The capsule of the spleen on the convex side was raised by a subcapsular hæmatoma which had ruptured through the capsule in two different areas. Otherwise the spleen appeared normal in size and texture.

PATHOLOGICAL REPORT.—Mr. D. M. Greig sent the following report of the microscopical details : " Well-marked Malpighian bodies with central artery. Thickening and hyalinization of wall of central artery. No very distinctive pathological appearances."

Etiology.—The etiology and mechanism of rupture of the apparently normal spleen have been well discussed by Susman.[2] The spleen ages early, and at 36 years 50 per cent of spleens show thickening and hyaline changes in the Malpighian bodies and capsule. These degenerative changes tend to rupture. Congestion of the portal vein and its radicles will force blood between the spleen and its investing capsule of peritoneum, giving a subcapsular hæmatoma. A slight degree of torsion of the splenic pedicle, which in a multiparous subject would be prone to twist, might cause the hæmatoma to burst.

SUMMARY.

1. A case of spontaneous rupture of an apparently normal spleen is recorded.

2. Only eleven authentic cases could be collected by Hamilton Bailey in January, 1930.

3. The etiology of rupture is problematical, but Mr. D. M. Greig, of the Royal College of Surgeons, Edinburgh, suggests in this case torsion of the splenic pedicle, which is feasible in a female of the maternal visceroptotic type such as the patient whose case is reported.

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RESULT OF TRANSPLANTATION OF URETERS MORE THAN A QUARTER OF A CENTURY AFTER OPERATION.

By P. R. ALLISON,
THE GENERAL INFIRMARY, LEEDS.

Transplantation of both ureters into the rectum for the relief of ectopia vesicæ is an operation which has been performed on many occasions. Some of the cases succumb either as a result of the operation or from ascending pyelonephritis a few months or years later. The case reported below has survived for twenty-seven years in a state of good general health, and is therefore of sufficient rarity and interest to be reported.

The patient, E. V. B., male, aged 7 years, was admitted to the General Infirmary at Leeds on July 21, 1905, under the care of Mr. Lawford Knaggs, suffering from incontinence of urine since birth as a result of ectopia vesicæ. His mother picturesquely ascribed the deformity to a fall from a dog-cart which she sustained during pregnancy. When the child was 11 months old he was taken to the Infirmary, where Mr. Knaggs performed a plastic operation on the anterior abdominal wall; the boy, however, developed measles, and in consequence of his poor general condition the wound failed to heal.

During his stay in hospital he cried so much that the presence of a hitherto unsuspected left inguinal hernia became apparent. On discharge he was supplied with an instrument, consisting of a cage strapped to the abdomen with a tube leading into a receptacle on one leg, and with this he existed until the age of 7, but of this period of his life he remembers very little.

The accidental breakage of the instrument caused his mother to consult Mr. Knaggs again, with the result that the patient was readmitted to hospital and the operation of transplantation of ureters was undertaken. The following is an abstract of the clinical notes made at that time (1905):—

On examination the upper two-thirds of the bladder are covered with thickened epithelium, whilst the lower part of the mucosa is raw and in a state of irritation. The urine can be seen dribbling from the ureteric orifices on to the raw area. The upper part of the penis is deficient, the testicles are not descended, and the pubic bones are separated by 2 to 3 in. The recti abdominis form a V-shaped hood to the bladder. A left inguinal hernia is present.

Operation was performed by Mr. Lawford Knaggs, on Aug. 1, 1905, under ether anæsthesia. The rectum was washed out. The bladder was first separated from its connections and from the peritoneum, leaving both ureters attached; the latter were splinted with catheters. Douglas's pouch was then separated from the rectum; the latter was pushed up by the fingers and a horizontal incision was made into it. The bladder was trimmed, leaving a portion with the ureters attached, which was drawn through into the rectum. The rectal aperture was closed by three sutures uniting the bowel wall and separating the ureters. The anal sphincter was dilated, a drainage tube inserted, and the suprapubic wound packed.

Following this operation the patient left Leeds and was not heard of for many years. Between the ages of 7 and 13 he went to school and played all

the usual games along with other children, but was, however, excused from the gymnasium. His general health was very good. About every two or three hours he passed per rectum a mixture of urine and fæces, and occasionally the intervals were as long as four hours. So long as he had the opportunity for evacuation at these times there was no incontinence, but if circumstances necessitated delay, there was considerable discomfort and even leakage. In addition to these liquid motions he passed a solid stool usually once a week.

From the age of 13 to 19 years he was lather boy and ran errands in a hairdresser's shop, after which he took a post as a lorry driver. This last occupation entailed the lifting of packages up to one and a half hundredweight and was indeed a very strenuous existence, yet he never felt any ill effects and was as competent and useful as his fellows. His day's work started at 7 a.m. and finished at 5 p.m., and at this time he was having a fluid evacuation every three to four hours and a solid motion every other day. In 1929 he married, but there have been no children.

His period of good health was only rarely interrupted by occasional colds, and in 1926 by an attack of lumbago which passed off in a few days. This consisted of an aching pain on both sides of the back, and was believed to have resulted from his heavy work; it was not associated with any pyrexia, sweating, or general ill health, so that it was probably not an acute renal infection. In 1930 he was off work for three days on account of a severe vomiting attack, but this appears to have been due to a dietetic upset and was not associated with any febrile symptoms or pain in the loins.

In July, 1931, he had his first serious set-back; he began to feel ill, had cold shivers, vomited, sweated, and had a raised temperature. His doctor diagnosed influenza and kept him in bed for a fortnight. The patient subsequently complained of weakness, malaise, loss of appetite, and a numb pain in the left thigh, which was present only on standing and disappeared on lying down. In this condition he was admitted to the hospital under the care of Dr. Watson.

On admission on Aug. 13, 1931, he looked pale and appeared to have lost weight, his pulse was 88, temperature (in the axilla) 97·5°, and respiration 24. The tongue was clean.

The abdominal wall was well healed anteriorly, but was weak and required the support of a belt. Both testicles were in the scrotum and a left inguinal hernia was present. The penis was small and deficient on the dorsum, where at the junction with the abdominal wall there was a small raw area discharging a little mucous secretion. The pubic bones were separated by 2½ in. There was tenderness and swelling in the left loin with some rigidity. The blood-count showed leucocytes 11,000 (neutrophil polymorphs 72 per cent, eosinophils 0·5 per cent, lymphocytes 21 per cent, monocytes 6 per cent). Blood-urea nitrogen was 26·1 mgrm. per cent.

On Aug. 17 the temperature was 99·6° and the pulse 96, after which there were frequent evening rises, on one occasion to 103°, with a pulse of 104.

An X-ray of the urinary tract showed a single calculus on the left side in a position corresponding to the pelvis of the kidney. Within a few days the swelling in the loin increased in size and became definitely fluctuant. On Aug. 25 the patient was transferred to the care of Mr. Flint, who incised the

loin and liberated a large quantity of foul-smelling pus. The abscess cavity was so large that its exact origin was difficult to determine, but, as neither stone nor kidney could be palpated, it was considered to be a large pyonephrosis.

On Aug. 20 a pyelogram was taken by means of intravenous injection of abrodil, and this showed a greatly hypertrophied kidney on the right side. The pelvis and calices were rather large, but the latter showed normal cupping, so that the increase in their size was presumably only in proportion to the degree of renal hypertrophy. No secretion of dye could be made out on the left side, which confirmed the suspicion that this kidney had been almost destroyed. Although the right ureter could be seen in parts the details of its structure and its opening into the rectum could not be determined. Sigmoidoscopy was not performed.

Following the operation the patient made rapid progress both in his general health and as regards the local condition. After discharging pus for six weeks the sinus in the loin almost healed except for a small quantity of serum, and on Sept. 30, 1931, he was discharged, apparently cured.

He says that when he returned home he felt so well that he wanted to go back to his work, but was advised to spend a few weeks breaking himself in gently. Four weeks later, however, he was quite suddenly taken ill and was readmitted to the hospital *in extremis*. At that time he was having repeated rigors and persistent hiccup; the wound in the loin broke down and discharged urine and pus, and the blood-urea nitrogen was 36·6 mgrm. per cent.

On the advice of Mr. Flint a large tube was inserted into his rectum in order to drain the bowel; urinary antiseptics and diuretics were administered, and on this treatment he gradually improved and was sent home to the care of his own doctor. From Dec. 1, 1931, until March, 1932, he remained in bed at home with the drainage tube in his rectum day and night, but by the latter date the sinus in the loin had healed completely, so he then discarded the tube during the daytime and started sitting up a little in the bedroom. In May, 1932, he had a femoral thrombosis in his left leg and had to return to bed, the leg being very swollen, cyanosed, and painful.

It was at this time that he discarded the rectal tube altogether, and the subsequent treatment has been directed against the condition of the leg, which still swells towards the end of the day. He is, however, up and walking about, and subjectively feels much fitter than he did during the two or three years preceding his recent illness. His appetite is very good indeed and he has gained weight so rapidly that a lumbar hernia has occurred through the loin incision; this, however, is adequately controlled by a modification of the abdominal belt which he has always worn. At present he can retain his motions for four hours, and curiously enough he passes a solid stool every day. His blood-urea nitrogen on July 5, 1932, was 24·3 mgrm. per cent.

Another very interesting thing which is difficult to explain is that the patient can now tell whether the motion he is about to pass is a fluid or a solid one, and he was never able to do this before his illness. He does not pass flatus except along with his usual evacuations.

I wish to thank Mr. Lawford Knaggs, Dr. Watson, and Mr. Flint for permission to publish this case.

ENCYSTED PEDUNCULATED HÆMATOMA IN THE PLEURAL CAVITY IN ARTIFICIAL PNEUMOTHORAX.

By J. B. CHRISTOPHERSON,

PHYSICIAN TO THE CITY OF LONDON HOSPITAL FOR DISEASES OF THE HEART AND LUNGS.

It seems worth while to record the following account of a rare and interesting surgical and anatomical condition—namely, an encysted hæmatoma with a twisted pedicle in the pleural cavity of a male aged 23, whose lung was artifically collapsed for pulmonary tuberculosis. The hæmatoma evidently was due to an incident or accident. The intercostal vessels had in my opinion been wounded when unsuccessfully exploring to withdraw pleural fluid which

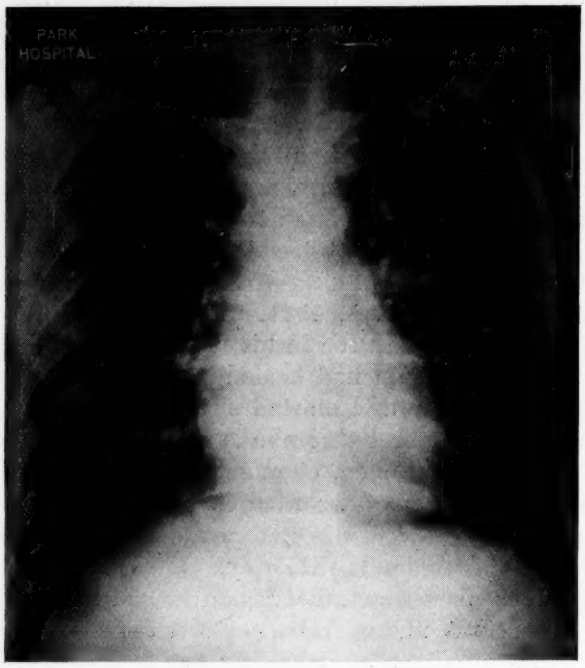

Fig. 317.—Jan. 13, 1932. Bilateral tuberculosis. Fair-sized cavity. Left apex seen below clavicle.

occurred in the course of artificial pneumothorax treatment. The hæmorrhage produced a subpleural tumour which, owing to the elasticity of the pleural membrane, became sausage-shaped, then, twisting on its long axis, acquired a pedicle and was a pedunculated cyst nine days later—with a twisted pedicle attached to the inner aspect of the thoracic wall. The accompanying skiagrams (*Fig.* 317–322) confirm my deductions. *Fig.* 317 was taken on the patient's admission to hospital on Jan. 13, 1932. It will be noted that these X-ray films are considerably clarified by the introduction

of lipiodol (*Fig.* 318, taken on May 15, about three weeks previous to the exploration on June 5 which caused the hæmatoma).

In the film taken on June 11, six days after the date of the wounding of the intercostal vessels by the exploring needle, there is to be noted, in the anterior-posterior film, a small triangular shadow marked A (*Fig.* 319). Then if one turns to the lateral view taken on the same day (*Fig.* 320), there is to be noted a round shadow wholly submerged in the pleural effusion. This was evidently the cyst, which was not present in the film taken on May 15 (*Fig.* 318) about three weeks before the date of the exploration with

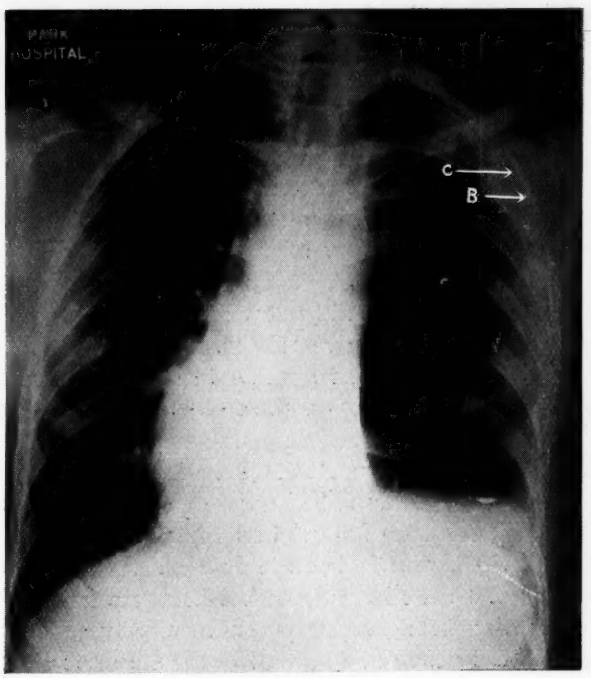

FIG 318.—May 15, 1932. Left artificial pneumothorax. Showing adhesions (3)—B and C to third and 4th ribs, another to dome; pleural effusion; lipiodol lies in pool unmixed with pleural fluid; some has adhered to thoracic wall and adhesions.

the needle and syringe. The four dots denoting the circular shadow were made by Dr. Sparks, Radiologist to Victoria Park Hospital.

On June 17 I did a thoracoscopy on the patient, dividing with the cautery three apical adhesions which were, I thought, causing the effusion; after completing the division of the special adhesions I introduced into the thoracic cavity, through the thoracoscopic puncture, a catheter, and, with the excellent and efficient Chandler two-way syringe, withdrew 14 oz. of pleural fluid.

With the fluid withdrawn I obtained with the thoracoscope a clear view of the lower end of the thorax and saw lying on the side of the interior of the thorax, posteriorly, a bluish-white body resembling a half-inflated balloon, with a twisted pedicle lying on, and the pedicle attached to, the

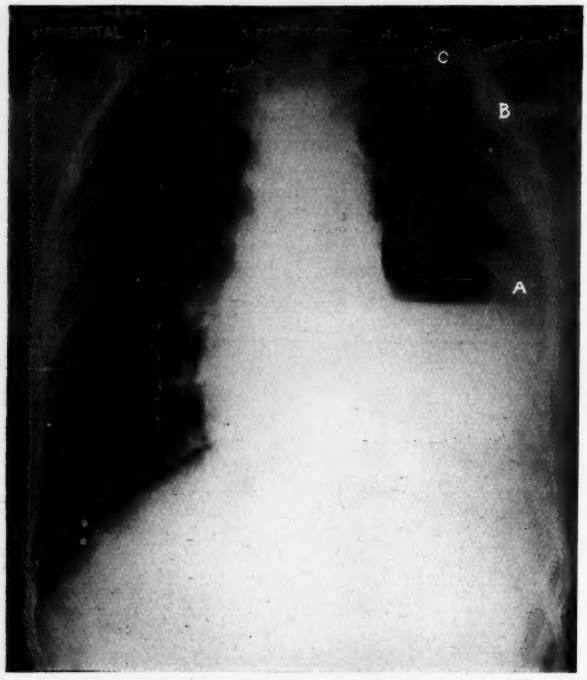

FIG. 319.—June 11, 1932. Skiagram taken after failure to evacuate fluid. Needle of exploring syringe was obstructed by cyst lying against posterior wall. A, Attachment of pedicle of cyst; B, C, Adhesions.

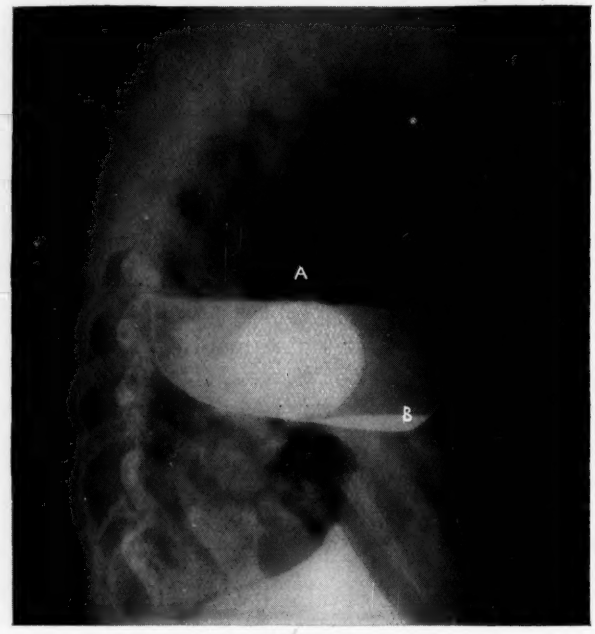

FIG. 320.—June 11, 1932. Lateral view. Loculated effusion. A, Base of stalk of adhesion. Globular mass attached to stalk and submerged. B, Lipiodol at bottom of fluid.

inner aspect of the thorax, I should judge tethered approximately where the needle had been used.

The cyst was evidently thin-walled, adapting itself to the shape of surrounding structures. This important point can be verified in the X-ray which I had taken immediately after the thoracoscopy (*Fig.* 321).

Finally, in the film (*Fig.* 322) made twelve days after the thoracoscopy operation (on June 29) the fluid was re-accumulating and the cyst had disappeared below the level of its surface, and so Miss Mickleburgh, the Radiographer at the City of London Hospital, Victoria Park, took the X-ray at about 5.0 p.m., the patient *lying on his side* in order to drain the fluid away from, and to isolate, the cyst. A very interesting film was obtained,

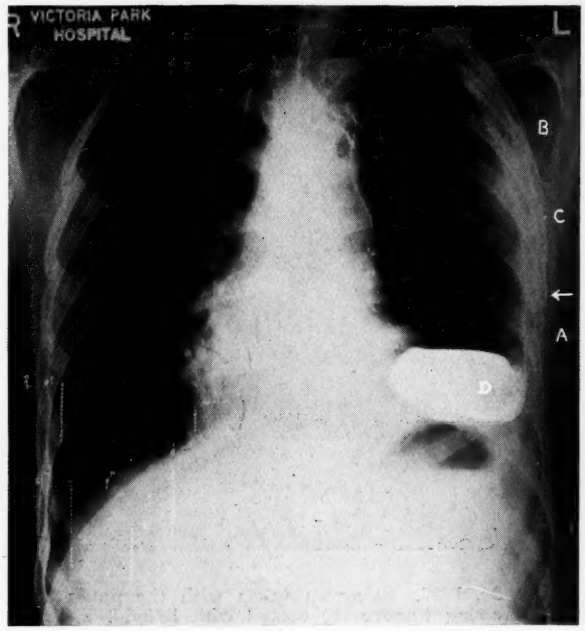

FIG. 321.—June 17, 1932. After thoracoscopy and division of adhesions by cautery. Soft purple tumour (D) with narrow long stalk attached to thoracic wall at A. B, Lung stump after division of two adhesions. C, Metal skin clip.

and attention is drawn to the now egg-shaped cyst, which had taken a brilliant coating of the lipiodol which I had introduced fourteen days previously (18 c.c.) for therapeutic reasons.

If this reproduction is viewed, as it was taken, in the horizontal position, a fluid level will be noticed situated above the diaphragm ; this is the pleural fluid displaced and lying over the mediastinum. Yet another fluid level is seen below the diaphragm ; this is the level of the gastric contents—tea, etc., with the gastric air-bubble above—the man having just finished his tea at 5.0 o'clock before being X-rayed.

It may be added that there was no pain, no rise of temperature, and no other symptom complained of attributable to the cyst.

The case appears to point: (1) To the desirability of avoiding the intercostal vessels by a knowledge of their relation to the ribs; (2) To the use of lipiodol for enhancing radiological pictures of the pleural cavity; (3) To the fact of the elasticity and extensibility of the pleural membrane; (4) Incidentally, one might add to the fact that lipiodol is inert in the pleural

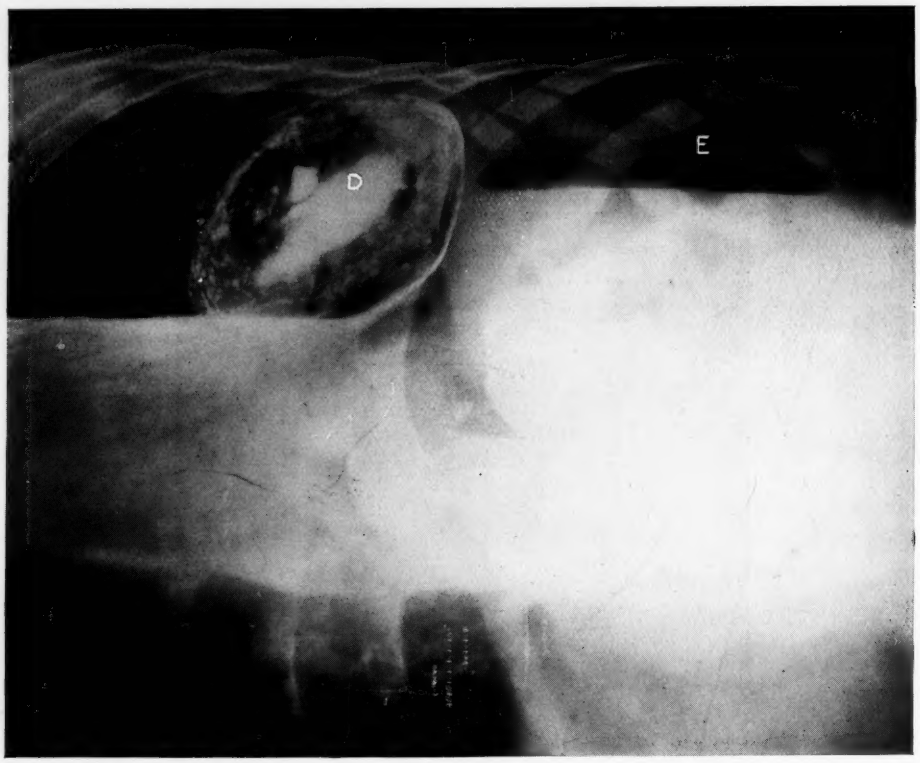

FIG. 322.—June 29, 1932. Skiagram taken with the patient lying on his side to displace fluid. Showing tumour (D) coated with lipiodol introduced on May 13 (18 c.c.). E, Gastric bubble.

cavity, that it takes a long time to absorb, that it does not mix with the pleural fluid, being an oleate, that it causes no inflammatory action in the pleura, or any action other than radiological—it is, in fact, therapeutically inert so far as the pleura is concerned.

Lipiodol is useful in ordinary radiology of the pleural cavity, also in stereoscopic radiology, and in thoracoscopy.

REVIEWS AND NOTICES OF BOOKS.

William Stewart Halsted. Surgeon. By Dr. W. G. MacCallum, with an Introduction by Dr. W. H. Welch. Demy 8vo. Pp. 241 + xvii. Illustrated. 1930. London: Oxford University Press. 12s. 6d. net.

The discovery of the practicability of general anæsthesia in the middle of the nineteenth century changed fundamentally the surgeon's outlook. Seemingly unlimited operative possibilities presented themselves to his mind. The rewards for daring and digital dexterity became tremendous. Dr. Halsted, in addition to being endowed with these qualities, possessed also the knowledge with which to implement them. Temptation lay before him. But for his fine, critical, and modest mind we might to-day remember him only as a figure that passed brilliantly through the operating-theatres of the eighties and nineties—a facile and ephemeral success. Instead, by guiding, and with personal discovery directing, the development of surgery he has made a permanent and unforgettable contribution to the welfare and happiness of human kind. In applying himself to the meticulously accurate surgery that strives continuously after a good physiological result he did much to protect the patient from the spectacular type of surgery, the disillusioning results of which are rapidly revealed by a study of the post-operative progress of its subjects. Dr. Halsted developed and taught a surgical technique as spectacular in its results as it was unspectacular in its performance.

But the founding of a great school of surgery expresses only a part of his activity. Every new and hitherto untried step taken in the operating-theatre was preceded, in so far as was possible, by minute and accurate clinical research upon animals in the laboratory. One of Dr. Halsted's happiest bequests to the students in Baltimore is the privilege they now enjoy of observing and taking part in this system of experimental study that he devised. In his early New York days he conceived ideas that were the basis of much fruitful research in that and later periods of his life. His block-dissection method in the treatment of cancer of the breast altered profoundly the prognosis in this affection. The operation he evolved for the cure of inguinal hernia has been of almost infinite benefit. So also have his discoveries in relation to local anæsthesia by the use of cocaine. His procedures in the treatment of aneurysm and in the treatment of acute infections of the gall-bladder are of established and recognized value. Dr. Halsted was one of the first surgeons in America to realize the enormous significance of Lister's discoveries. In face of the scepticism and unbelief of his colleagues he followed rigorously the antiseptic routine, and so, by dispelling the fear of the dreaded post-operative suppuration, completed the emancipation of the American surgeon that had been begun by the discovery of general anæsthesia. Not until Germany, and America under Halsted, had been for some time following the principles laid down by Lister did the English appreciate their value. Here, as always, Dr. Halsted was closely conversant with any surgical advance made in Germany, for whose surgeons he had a great and reciprocated respect.

This interest in German work and discovery dated from the two years Dr. Halsted spent in his youth as a post-graduate student in Germany, Austria, and France. During these years he made many friends. The high regard in which he held his foreign friends is proved by the generous way in which he helped to relieve the circumstances of those amongst them that were placed in difficulties after the war. His anxiety about the well-being of other surgeons exemplifies his concern for the well-being of surgery itself. One of Dr. Halsted's finest qualities was his

quick and unselfish appreciation of anything new and significant in the work of others. In this way, by practising and interpreting the work of others, he performed some of his greatest services to surgery. Pioneer, investigator, and distinguished teacher, he ranks with the great surgeons of his period.

History of Scottish Medicine. By JOHN D. COMRIE, M.A., B.Sc., M.D., F.R.C.P., Physician to the Royal Infirmary, Edinburgh, etc. Second edition. Two volumes. Large 8vo. Pp. 852, with 2 plates and 404 illustrations. Published for the Wellcome Historical Medical Museum. 1932. London: Baillière, Tindall & Cox. 50s. net.

THESE volumes, which form the latest addition to the Wellcome series of medical historical monographs, are of extreme value. They are written by Dr. Comrie, who is Physician to the Royal Infirmary of Edinburgh and Lecturer on the History of Medicine in the Edinburgh University. They have the merit of being exhaustive for the country with which they deal. Scotland has long occupied a leading position both in the teaching and practice of medicine. A succession of great teachers in the eighteenth century brought its schools into prominence and attracted students from England, from Ireland, and more especially from America. Young men of talent who would have gone to Paris to learn anatomy and to Leyden for their clinical teaching now fared equally well at Edinburgh—or even better, for the language difficulties were less. The foreign tour, which was universal in the seventeenth century, became less and less frequent without loss of the advantages which used to be gained from it. The teaching of Boerhaave and Haller bore splendid fruit, for the earliest physicians like Archibald Pitcairne, Cullen, and James Gregory had been themselves taught at Leyden and Göttingen. Midwifery, too, had always had a special attraction for the Scottish doctors because the midwives were untrained, very ignorant, and few in number. The work of Smellie therefore appealed especially to them and the teaching was of a high order. It was otherwise with surgery. The country was poor, the means of transit were bad, and the constant clan warfare made travelling dangerous. Surgery, as we understand it, was only possible in Edinburgh and Glasgow, for even at Aberdeen there was no systematic teaching of the subject until late in the last century. It was hampered, too, by its constant alliance with anatomy, and the Professor of Anatomy and Surgery was usually more interested in the dissections which he practiced daily than in the operations which he never undertook. Clinical teaching was given as early as 1748 in medicine by those who had been taught at Leyden, and an attempt was made to hold classes in clinical surgery in 1769, but it was premature, and no regular classes were held until 1803.

Dr. Comrie gives some interesting accounts of the surgeons practising in Edinburgh. He says "one of the best known of the eighteenth-century surgeons was Alexander Wood (1725–1807) known to his contemporaries as 'Lang Sandy Wood', and greatly respected for his dexterity in practice, which did much to raise the reputation of the surgical department in the Royal Infirmary, as well as beloved for his amiable social qualities." He was represented by John Kay, who drew the well-known series of portraits of Edinburgh's worthies, in wig and cocked hat with an umbrella under his arm in allusion to the fact that he was the first person in Edinburgh to make use of that article. At a time when personal peculiarity was widely affected in the capital Wood specially distinguished himself by going to see his patients accompanied by a pet sheep and a raven. The life of a surgeon in Scotland was not usually cast in such pleasant places. The customs of the period were primitive and curious. A surgeon would start on Monday caparisoned for the week with drugs and surgical appliances and would not return home until Friday. To obviate the dangers of travelling by night he carried a lantern fastened by a strap above his knee. The bull's-eye of the doctor's lantern was often signalled on moonless nights, heralding the comforting assurance of an obstetric deliverance. His regularity in his rounds vied with the carrier of His Majesty's mails; the saddle-bags of the one and the surgical accoutrements of the other were similarly horsed so that the laird scanning the road could say, "It's either the doctor or the post that's

coming." By such sidelights Dr. Comrie lightens the history and makes it most readable. He has, too, been at great pains to select lifelike portraits of the men he mentions, and the whole book is illustrated lavishly. Chapter and verse are given for all the statements, so that it is easy to test their accuracy and obtain more detailed information. There is a good index and the book is excellent from beginning to end.

Chirurgische und konservative Kosmetic des Gesichtes. Edited by Dr. LEANDER POHL (Vienna). Large 8vo. Pp. 383 + viii, with 445 illustrations and 3 coloured plates. 1931. Berlin and Vienna: Urban & Schwarzenberg. Paper covers, RM. 25; bound, RM. 28.

THIS book will appeal to few practitioners in this country, for as yet no specialty entirely devoted to cosmetic medicine has appeared in England. Nevertheless, that the subject is considered of growing importance on the Continent is evidenced by the recent increase in German and French literature devoted to it, and by the fact that this latest addition edited by Dr. Pohl is issued under the ægis of Professor von Eiselsberg. It represents a collaborative attempt to group together all those medical and surgical affections of the face which may be described as of cosmetic importance. Thus it invades boldly fields which in this country have hitherto belonged almost exclusively to the physician, the dermatologist, the roentgenologist, the dentist, the general surgeon, and finally to the already highly specialized plastic surgeon.

It contains a great deal of interesting material and much that can be obtained only from scattered sources, and for that reason is a valuable contribution. The various sections, however, suffer from a certain unevenness both in quality and completeness which rather detracts from the usefulness of the book as a whole.

Pohl contributes an interesting study of facial proportions and their variations from youth to old age. A complex analysis of facial components and their combinations both in profile and full face enables him to separate individuals into definite facial groups. For this purpose he uses a measuring instrument and a photographic recording device. Facial appearance characteristic of a wide variety of general diseases is discussed by Kollert, while the more local affections of the face are dealt with by Stein. Holzknecht contributes an account of his treatment of hypertrichosis by X rays, a method no longer in vogue in England. The plastic surgical treatment of defects of the cheeks, nose, mouth, ears, and eyelids is dealt with by Frisch, Safar, and Ehrenfeld. This section follows conventional lines and might have been materially improved by better diagrams and illustrations. Plastic surgery is dominated largely by mechanical difficulties and lends itself particularly well to graphic and pictorial illustration. Pichler deals with lesions of the lower jaw requiring resection, bone-grafting, or the use of prosthetic appliances. The section devoted to micrognathism and macrognathism is of considerable interest.

The chapter on cleft lip and palate is rather disappointing. Neither Brophy's nor Mirault's operation warrants serious consideration in an up-to-date discussion of this subject. No mention is made of the excellent contributions of Vaux, Ritchie, or Davis, nor of the opinions so often expressed by Gillies, Fry, and Wardill on the necessity and means for obtaining apposition between the soft palate and the posterior pharyngeal wall for functional success. Another omission is the treatment of secondary defects of the lip following primary closure—a matter of paramount cosmetic importance and a type of defect frequently encountered in the practice of plastic surgeons. The book concludes with a section devoted to malformations, malpositions, and diseases of the teeth, and their treatment by medical and orthodontic means.

The volume covers so wide a range of subjects that one cannot escape the feeling that the field is too comprehensive ever to be considered as one specialty or to be efficiently handled by one person. On the other hand, it indicates the supreme importance of close co-operation between medical, surgical, and dental experts in the cosmetic treatment of facial affections.

The Acute Abdomen. By C. H. FAGGE, M.S. Lond., F.R.C.S., Surgeon to Guy's Hospital. Pocket Monographs on Practical Medicine. F'cap 8vo. Pp. 92 + viii. 1932. London: John Bale, Sons & Danielsson, Ltd. 2s. 6d. net.

THIS volume is one of the Pocket Monographs on Practical Medicine, and is a very small book dealing with a large and difficult subject. Mr. Fagge, however, manages to give the reader a wealth of information based on his extensive practical experience. The book is divided into three parts. In the first the various symptoms met in an acute abdominal catastrophe are outlined and their significance and varying importance discussed. The next part considers the differential diagnosis of the individual lesions, and the final part the principles of treatment. On debatable subjects, such as the treatment of acute appendicitis, Mr. Fagge states his views from the aspect of pathology and practical experience. We cannot agree, however, that the point of tenderness in acute appendicitis does not alter with the different positions of the appendix.

The whole of the matter is set out in a perfectly clear and simple manner, and the book can be read in a few hours under armchair conditions. There are no illustrations. House surgeons and many others will benefit by a perusal of this volume, and they will not go far wrong if they adhere to the author's advice but at the same time understand the basis of his reasoning.

A Clinical Study of the Abdominal Cavity and Peritoneum. By EDWARD M. LIVINGSTONE, B.Sc., M.D., Associate Visiting Surgeon, Bellevue Hospital, New York. Imperial 8vo. Pp. 866 + xxii, with 372 illustrations. 1932. New York: Paul B. Hoeber, Inc. $15.00.

IN this book the author has compiled a comprehensive review of abdominal symptoms and signs with special reference to the embryological, anatomical, and physiological factors concerned. The work is intended for students or graduates, and by its arrangement can easily be used to a good purpose by either.

Of the many sections, those dealing with the nervous mechanism of the abdomen —both motor, sensory, and sympathetic—are perhaps the most striking; these chapters are freely illustrated with useful and informative charts and diagrams. In the final chapter on the clinical interpretation of viscero-sensory phenomena a full and careful estimation is given of the value of the various sensory skin manifestations in intra-abdominal affections. As a work of reference the book will undoubtedly serve a useful purpose.

Diagnosis in Joint Disease: A Clinical and Pathological Study of Arthritis. By NATHANIEL ALLISON, M.D., F.A.C.S., Professor of Surgery, University of Chicago, etc.; and RALPH K. GHORMLEY, M.D., Associate in Orthopedic Surgery, Mayo Clinic, etc. Large 4to. Pp. 196 + xii. Illustrated in colour and in black-and-white. 1931. London: Humphrey Milford. 52s. 6d. net.

THIS monograph deals with all the principal forms of arthritis—tuberculous, traumatic, loose bodies, pyogenic, rheumatoid, and osteo-arthritis. In each section there are many clinical histories of typical cases, radiographs of early, moderate, and late stages of the disease, and histological pictures of the tissues involved. This method of presentation gives a very clear and complete account of each disease, and the accuracy and beauty of the illustrations call for great praise. The only criticism of the illustrations which we can make is that the reproductions of colour photographs are disappointing, because they give no clear definition of the conditions depicted. This, however, only applies to the frontispiece and to *Plate VII*. The terms 'rheumatoid arthritis' and 'osteo-arthritis' are avoided, the former being chiefly covered by the phrase 'proliferative arthritis', and the latter by that of 'degenerative arthritis', but in each case the illustrative figures give an excellent picture both of the joints and the tissues which are affected.

The book as a whole will form an admirable source of reference.

Minor Surgery of the Urinary Tract. By H. C. BUMPUS, Jun., with chapters on Caruncles by J. L. CRENSHAW, and on Postoperative Care by A. L. CLARK. 8vo. Pp. 124, with 57 illustrations. 1932. Philadelphia and London: W. B. Saunders Company. 15s. net.

THIS book is one of the Mayo Clinic monographs, and gives in a clear and concise manner descriptions of minor surgical procedures as practised in the genito-urinary department of that institution. It opens with a short account of general considerations such as anæsthesia, preparation of the patient, etc. Next are given the details of the neat and effective method of dealing with that painful lesion, the urethral caruncle. Perhaps the most interesting chapter is that concerned with hypertrophy of the prostate gland. Bumpus here describes his modification of the Braasch 'punch' operation for the removal of the obstructing portion of the gland. This endoscopic method of treatment of the enlarged prostate may now be said to have passed from infancy to adolescence, and in the future is obviously destined to take an increasingly prominent place in the surgery of this condition.

Two interesting and rare affections of the bladder—namely, interstitial cystitis (Hunner's ulcer) and alkaline phosphatic cystitis—are discussed in considerable detail. The manipulative extraction of stones in the ureter is a subject which is treated with thoroughness, the several methods of treatment employed being described fully. With most of the post-operative care suggested we are familiar, but an unusual feature of this chapter is that on the use of a ketogenic diet as a means of acidifying the urine. We imagine that this method of altering the pH of the urine, as an adjuvant in the treatment of infections of the urinary tract, is still in the experimental stage. However, if it can be proved definitely to be of use in cases of chronic pyelonephritis, then it is surely worthy of consideration.

Amongst surgeons in general, and in particular amongst those who are interested in surgery of the urinary tract, this small monograph is likely to prove deservedly popular.

Diseases of the Kidney. By W. GIRLING BALL, F.R.C.S., Surgeon to St. Bartholomew's Hospital; and GEOFFREY EVANS, M.D. (Cantab.), F.R.C.P. (Lond.), Physician, with charge of Out-patients, St. Bartholomew's Hospital. Royal 8vo. Pp. 424 + viii, with 159 illustrations and 8 coloured plates. 1932. London: J. & A. Churchill. 36s. net.

SINCE Rayer published his omniscient book, *Les Maladies des Reins*, the subject has become so vast and complicated that there must be few indeed who would venture to write a monograph which should bring up to date our knowledge of the diseases of the kidney from both the medical and surgical points of view. This volume is a noteworthy attempt to do so within the limits of a book of reasonable size; the authors are a surgeon and a physician at St. Bartholomew's Hospital, and, with the wonderful traditions of that ancient hospital to inspire them, we expect a monograph which will speedily become a classic.

The book is well bound and beautifully printed, and the numerous illustrations are delightful and a real help in the elucidation of the text; the latter, as one would expect of these two authors, is plain and straightforward, and most singularly free from printer's errors.

The chapters on movable kidney and tuberculosis of the kidney struck us as particularly good; under the former, we are relieved to find that the authors do not look on the occasional fixation of a movable kidney as a surgical misdemeanour, and their account of tuberculous disease of this organ is one of the best we have read.

There are one or two rather surprising omissions; we can find no mention of either hydatid disease or of actinomycosis, though both are well recognized, if very uncommon, affections of the kidney. The description of oxaluria would have been more complete if the question of the excessive flatulence which sometimes accompanies the attacks had been discussed. This omission is the more glaring since the best account of this syndrome was published by one of the authors' colleagues in the *Transactions* of the Medical Society of London. He gave an account of a house surgeon whose intestinal symptoms were so marked that a

laparotomy for intestinal obstruction was done; his condition after this operation was unchanged, and a second operation was contemplated, but, at a consultation of the Staff, it was found that the first examination of the urine had been wrongly reported and that it contained a good deal of blood and was full of oxalates.

Bright's disease is very thoroughly discussed, a chapter of nearly one hundred pages being devoted to it; we have read it through carefully, and our conclusion, owing perhaps to surgical prejudice, is that this disease has become so complicated as to be almost incomprehensible.

These are, however, very minor defects, and we can most heartily commend the book to our readers.

Intracranial Pyogenic Diseases. A Pathological and Clinical Study of the Pathways of Infection from the Face, the Nasal and Paranasal Air-cavities. By A. LOGAN TURNER, M.D., LL.D. Edin., Hon. F.R.C.P.E., F.R.S.E., F.R.C.S.E., Consulting Surgeon, Ear and Throat Department, Royal Infirmary of Edinburgh; and F. ESMOND REYNOLDS, M.D. Edin., D.T.M.&H. Camb., M.R.C.P.E., Lecturer on Neuropathology, University of Edinburgh. Royal 8vo. Pp. 271 + xx, with 82 illustrations. 1931. Edinburgh: Oliver & Boyd. 12s. 6d. net.

THIS volume, inscribed as it is "to the pioneer work of Sir William Macewen on intracranial infection", constitutes a fitting companion to that great surgeon's well-known classic, published in 1893, under the title of *Pyogenic Diseases of the Brain and Spinal Cord*. It embodies the results of some ten years' methodical and painstaking research into the pathways of intracranial infection, correlating the clinical and pathological observations in a series of 55 cases. In most of these the primary septic lesion was connected with the nasal and paranasal air cavities. In passing it may be remarked that the authors have rendered a service to clearness of nomenclature by rejecting the term 'sinus' as applied to the air-spaces in the bones. Throughout the book they designate these as 'air cavities', and reserve the word 'sinus' for describing the venous intradural spaces.

Of the 55 cases, the primary intracranial complications were leptomeningitis in 24, cavernous sinus thrombosis in 21, and cerebral abscess in 4. The remaining 6 cases were examples of generalized hæmic infection or pyæmia.

The beginnings of cerebral abscess in the authors' cases are clearly shown to have been retrograde thrombosis of veins followed by infection of the perivascular spaces.

Septic thrombosis of the cavernous sinuses occupies a large part of the book. These cases are divided into 'fulminating' (5), 'acute' (3), and 'restrained' (13). The mechanism of the ocular symptoms is fully discussed. Of the 21 patients, 1 recovered. In this case the primary focus was aural disease on the left side, with unilateral cavernous sinus thrombosis on the right side. There seems to be little ground for believing that in this instance the thrombosis was of a septic nature.

The book is excellently produced and well illustrated, 21 of the 82 pictures being beautifully coloured.

The Use of Lipiodol in Diagnosis and Treatment. By J. A. SICARD, Late Professor in the Faculty of Medicine, Paris, and J. FORESTIER (Aix-les-Bains). Royal 8vo. Pp. 235 + x, with 50 illustrations. 1932. London: Humphrey Milford, Oxford University Press. 16s. net.

The French edition of this work appeared in 1928. It deals chiefly with the diagnostic applications of lipiodol, and the various methods of its use are given in great detail. Possibly its utility in the localization of spinal tumours and other causes of spinal compression is overestimated, whilst the objections to its use in these cases are, though frankly discussed, minimized. Little, for instance, is made of the root pains and sphincter disturbances which sometimes follow intrathecal injection. Possibly such serious ill effects have been due to the use of lipiodol which has undergone decomposition. The authors insist strongly against the use of lipiodol which no longer possesses its original pale amber colour.

The account of the therapeutic uses of lipiodol occupies but fifteen pages. It has been employed for pain in various positions and of varying causation; and also for such conditions as chronic arthritis, tuberculous abscess, and bronchiectasis. The therapeutic effects do not appear to be such as to arouse enthusiasm.

It is rather surprising that of the 231 pages, 39 are devoted to bibliography, and that these pages contain references to no fewer than 708 papers relating to the subject.

The reproductions of the radiograms are greatly superior to those in the French edition.

St. Bartholomew's Hospital Reports. Edited by Sir THOMAS HORDER, Bart., K.C.V.O., RONALD G. CANTI, WILFRED SHAW, CHARLES F. HARRIS, J. PATERSON ROSS, R. C. ELMSLIE, W. GIRLING BALL, and GEOFFREY EVANS. Vol. LXIV. Demy 8vo. Pp. 231 + xxv. Illustrated. 1931. London: John Murray. 21s. net.

WHEN reviewing recent volumes of this series, we have had occasion to dwell favourably upon the peculiar happy notes which have appeared over the initials of present members of the staff in commemoration of the careers of their old teachers. Dr. Barris's obituary notice of Sir Francis Champneys is in no particular below the customary high level. It draws attention to Champneys' many-sidedness without unduly obscuring his prominence as a gynæcologist. It is sad to reflect how often one fails to glean the interest of one's acquaintances until their obituary notices appear. It may be news to many to read of Champneys' reputation as a learned and admirable musician.

Professor H. H. Woollard's paper on spastic paralysis contains a clear and up-to-date account of work on the physiology of the sympathetic system and the way in which it has been applied to the surgery of spastic paralysis.

J. P. Hosford writes very briefly on the treatment of common fractures. It is to be hoped that in a later volume he will write in more detail of his experience in the use of Kirschner's wire for axial traction.

Ralph Phillips contributes a very long and painstaking study of buccal cancer, which was a thesis approved for the degree of Master of Surgery in the University of London. Perhaps his chief conclusion is a corroboration of the common belief that the histological appearance bears a well-marked correlation with the clinical course of cancer.

BOOK NOTICES.

[*The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest for our readers.*]

Rose Research in Lymphadenoma. By Sir THOMAS HORDER, Bart., K.C.V.O., M.D., F.R.C.P.; M. H. GORDON, C.M.G., C.B.E., M.A., D.M., F.R.S.; KENNETH STONE, D.M., M.R.C.P.; LAWRENCE P. GARROD, M.A., M.B., M.R.C.P.; E. R. CULLINAN, M.D., B.S., M.R.C.P.; and B. D. PULLINGER, M.D., B.S. Crown 4to. Pp. 136 + viii, with 98 illustrations in colour and in black-and-white. 1932. Bristol: John Wright & Sons Ltd. 21s. net.

The Science and Practice of Surgery. By W. H. C. ROMANIS, M.A., M.B., M.Ch. (Cantab.), F.R.C.S. (Eng.), F.R.S. (Edin.), Surgeon and Lecturer on Surgery, St. Thomas's Hospital, etc.; and PHILIP H. MITCHINER, M.D., M.S. (Lond.), F.R.C.S. (Eng.), Hon. Surgeon to H.M. the King, etc. Fourth edition. Royal 8vo. In two volumes. Vol. I, General Surgery. Pp. 865 + x, with 373 illustrations. Vol. II, Regional Surgery. Pp. 1045 + x, with 326 illustrations. 1932. London: J. & A. Churchill. 14s. net per vol.

Treatment of Fractures in General Practice. By W. H. OGILVIE, M.D., M.Ch., F.R.C.S. F'cap 8vo. Vol. I, Pp. 108 + viii; Vol. II, 109–180. Illustrated. 1932. London: John Bale, Sons & Danielsson Ltd. 2s. 6d. each.

The Relative Value of Radiotherapy in the Treatment of Cancers of the Upper Air-passages. University of London Semon Lecture. By W. Douglas Harmer. M.A., M.B., M.C. (Cantab.), F.R.C.S. Demy 8vo. Pp. 85 + vi. Illustrated. 1932, London: John Murray. 6s. net.

Acromegaly. By F. R. B. Atkinson, M.D., C.M. (Edin.), with a Foreword by Sir Arthur Keith. Demy 8vo. Pp. 260 + x, with 3 plates. 1932. London: John Bale, Son. & Danielsson Ltd. 21s. net.

Kleine Chirurgie. By Prof. Dr. Hans Kurtzahn (Königsberg i. Pr.). Second edition. Large 8vo. Pp. 462 + viii, with 167 illustrations. 1932. Berlin and Vienna: Urban & Schwarzenberg. Paper covers, RM. 13.50; bound, RM. 15.

St. Bartholomew's Hospital Reports. Edited by Sir Thomas Horder, Bart., K.C.V.O., Ronald G. Canti, Wilfred Shaw, Charles F. Harris, J. Paterson Ross, R. C. Elmslie, W. Girling Ball, and Geoffrey Evans. Vol. LXV. Large 8vo. Pp. 1–317, with Index of Vols. XLI–XLV. Illustrated. 1932. London: John Murray. 21s. net.

A Synopsis of Surgical Anatomy. By Alexander Lee McGregor, M.Ch. (Edin.), F.R.C.S. (Eng.), Lecturer on Surgical Anatomy, University of Witwatersrand, etc., with a Foreword by Sir Harold J. Stiles, K.B.E., F.R.C.S. (Edin.). Crown 8vo. Pp. 609 + xvi, with 606 illustrations. 1932. Bristol: John Wright & Sons Ltd. 17s. 6d. net.

Erdmann's Clinics. Excerpts selected from the Clinics of John F. Erdmann, M.D., F.A.C.S., Professor of Surgery in Colombia University. Edited by J. William Hinton, M.D., F.A.C.S., Associate Professor of Surgery, New York Postgraduate Medical School. Large 8vo. Pp. 315 + vi, with 39 illustrations. 1932. Philadelphia and London: W. B. Saunders Company. 22s. 6d. net.

Practical Anatomy by Six Teachers. Edited by E. P. Stibbe, F.R.C.S., Senior Demonstrator in Anatomy, London Hospital Medical School. Demy 8vo. Pp. 719 + xii, with 337 illustrations. 1932. London: Edward Arnold & Co. 30s. net.

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